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OPERATIVE ORAL SURGERY

THE GIANT-CELL TUMOR

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GENUINE giant-cell tumors occurring in the maxilla or the mandible have caused much confusion and controversy. This entity is not as common in the jaws as many in the profession believe. A brief review of the literature and a case report may be of interest.

In 1940, Jaffe, Lichtenstein, and Portis[1] attempted to define what should be regarded as a genuine giant-cell tumor of bone. This is a distinct neoplasm arising from the non-bone-forming supporting connective tissue of the marrow, which can be readily identified on the basis of its histologic appearance. Histologically, the tumor is composed of a vascularized network of spindle-shaped or ovoid stromal cells, which is regularly and rather heavily interspersed with multinucleated cells (apparently syncytial stromal cells) as an integral part of the histologic pattern. It became evident to Jaffe and his colleagues that a genuine giant-cell tumor should be completely divorced from its "variants," with which it has been (and, to some extent, still is) confused.

According to Jaffe,[2] these "variants"—the fibromas, osteofibromas, xanthofibromas, solitary bone cysts, and lesions representing organization of hemorrhagic extravasations—have little in common clinically and anatomically with the genuine giant-cell tumor and little in common with each other except for a uniformly favorable prognosis.

Lichtenstein,[3] in his book entitled *Bone Tumors,* states that it is not too uncommon for localized rarefied defects resulting from hyperparathyroidism to be confused with genuine giant-cell tumors, so that attention is diverted from a search for the offending parathyroid adenoma and valuable time is thereby lost.

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In their book entitled *Tumors of Bone*, Geschickter and Copeland[4] write:—"... excluding the epulis of the alveolar border, the records of the surgical pathologic laboratory of Johns Hopkins Hospital extending over a period of 35 years showed only 22 cases of giant-cell tumor occurring in the head; 6 in the maxilla; 14 in the mandible; 2 in the temporal fossa." These authors also feel that the occurrence of a giant-cell tumor in the skull follows the distribution of the cartilaginous centers of ossification, supporting the view that these tumors are related to the normal process of ossification via cartilage. The development of giant-cell tumors of the alveolar ridge (giant-cell epulis), they state, may be one of the early signs of hyperparathyroidism.

Coley[5] examined 124 cases of giant-cell tumors and found none in the upper and lower jaws. Thoma[6] presents an excellent summary of views and cases in this category.

Fig. 1.—Clinical photograph showing mass in left maxilla between canine and first premolar.

CASE REPORT

A 35-year-old white woman was admitted for the second time to Montefiore Hospital in October, 1955, with a chief complaint of left flank pain radiating across the abdomen. This pain had been present for one week.

The history of the present illness revealed that the patient had complained of intermittent pain, which was more severe during urination. The patient also stated that she had difficulty in urinating and that the urine was exceptionally dark in color.

The patient's past medical history revealed that she was seen as an outpatient in March, 1946, when a diagnosis of chromophobe adenoma of the pituitary gland was made. Irradiation was recommended, and the prescribed course of therapy was carried out. The patient was again seen at this hospital in November, 1953, when she was admitted with a diagnosis of left ureterolithiasis, hydro-ureter, and hydronephrosis. She passed a stone on this admission. In May, 1955, the patient was seen in the genitourinary clinic, where a diagnosis of renal calculi was made. In July, 1955, during a follow-up examination by the radiotherapist, the patient complained of a swelling in left maxilla and was referred to the dental service.

A clinical examination at that time revealed a circumscribed, indurated mass, about 1 cm. in diameter, located in the left maxilla between the canine and the first premolar. The mass appeared blue-purple in color and had a central area of necrosis. A clinical diagnosis of central giant-cell tumor was made (Fig. 1).

A roentgenographic examination revealed a radiolucent, circumscribed area between the roots of the canine and the first premolar. The roots of both teeth appeared to be displaced by the mass (Fig. 2).

Under local anesthesia the tumor mass was excised in toto, and the canine and first premolar were extracted, since they were involved in the mass and exhibited excessive mobility. The specimen was submitted to the pathologist, who returned a microscopic diagnosis of giant-cell tumor of bone (Figs. 3 and 4).

The physical examination at the patient's second admission to Montefiore Hospital revealed a well-developed, obese white woman. The thyroid was normal to palpation. The remainder of the physical examination was essentially negative.

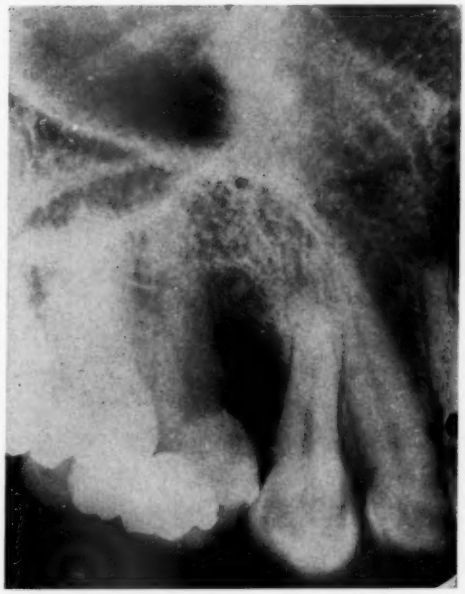

Fig. 2.—Roentgenogram showing radiolucent area in maxilla between canine and first premolar.

Laboratory examination of the patient's urine was within normal limits. Blood chemistries revealed a serum calcium of 15.1 mg. per 100 c.c., a serum phosphorus of 2.0 mg. per 100 c.c., and an alkaline phosphatase of 4.8 mg. per 100 c.c.

In view of the laboratory findings and the history of repeated renal calculi, the patient was examined for a parathyroid adenoma by the metabolic service. She showed a marked hypercalcemia on a low-calcium diet. Accordingly, in October, 1955, the neck was explored for a possible parathyroid adenoma. Two adenomas were found—one at the right lower pole and one at the left lower pole of the thyroid. Postoperatively, the patient did well. The blood chemistries returned to within normal limits.

CONCLUSION

It is suggested that any lesion of the jaws which is diagnosed microscopically as a giant-cell tumor be further investigated. The genuine giant-cell tumor is a rare entity. Hyperparathyroidism should be ruled out through

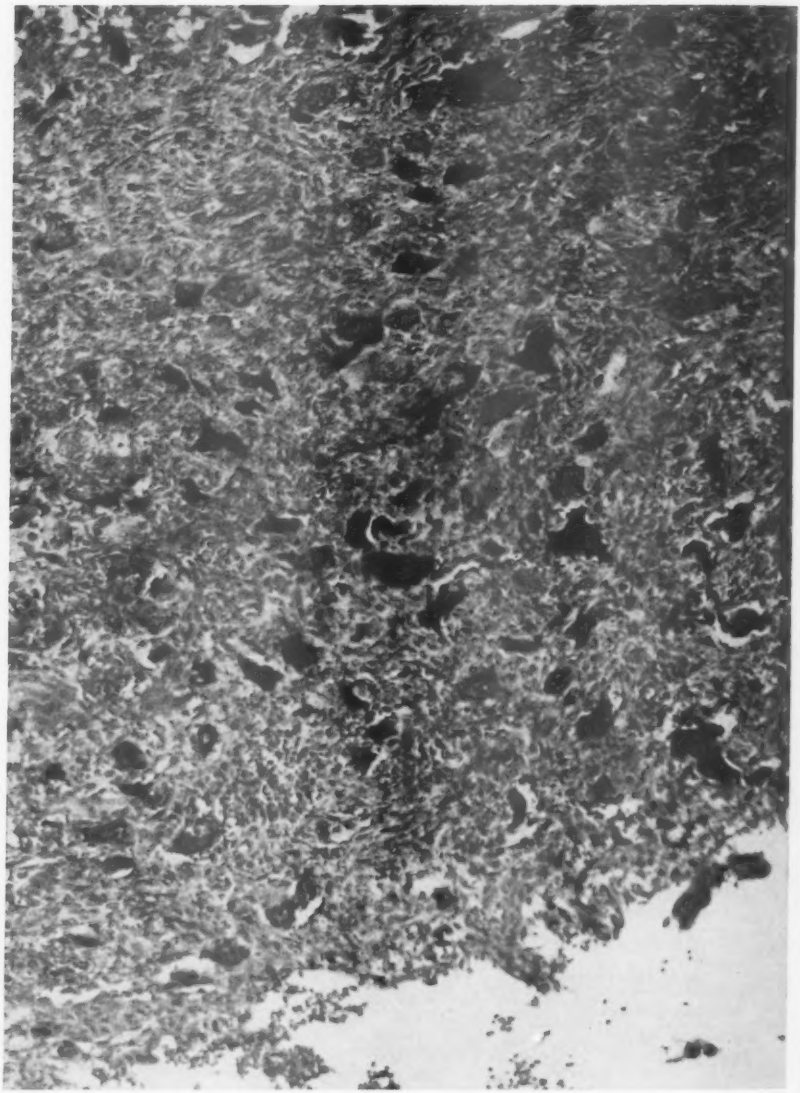

Fig. 3.—Photomicrograph of tumor, magnified 150 times, showing giant cells in a connective tissue stroma.

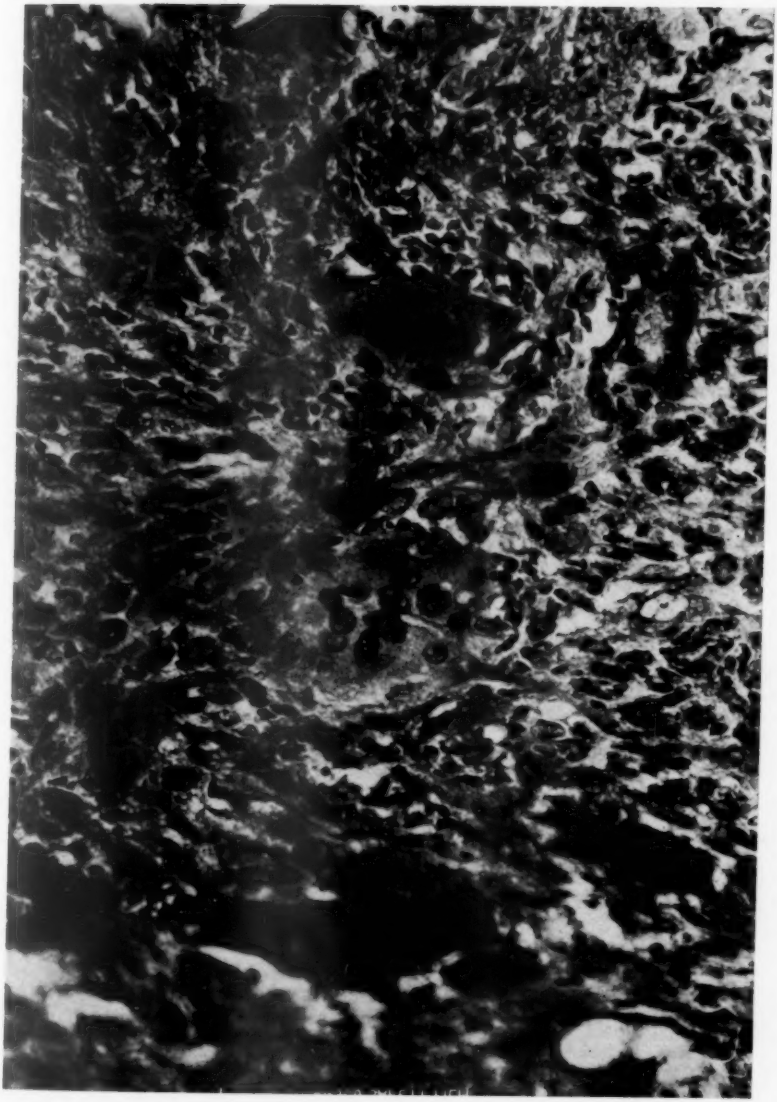

Fig. 4.—Photomicrograph of tumor, magnified 450 times, showing giant cells in a connective tissue stroma.

blood chemistry studies, even though no other radiolucent areas exist in the jaws. There also exisits a clinical relationship between renal calculi and hyperparathyroidism.[7]

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PAROTID ABSCESS

Report of a Case

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FASCIAL PLANE abscesses are uncommon today because of the widespread use of antibiotics. Therefore, we feel that the following case should be of interest.

CASE REPORT

A 42-year-old white American dairy farmer was referred by his dentist to the oral surgery service of the Ohio Valley General Hospital.

Chief Complaint.—The patient stated that he was suffering from pain and extreme swelling of the left jaw and that for the past three weeks he had not been able to open his mouth enough to chew food.

History of Present Illness.—Three weeks prior to admission, the patient's upper left second and third molars had been extracted under local anesthesia. On the third day following the extractions, pain, swelling, and trismus developed, producing a false ankylosis, and the patient was unable to masticate food. These symptoms became progressively worse until the time of the patient's admission to the hospital. During this period supportive treatment and antibiotic therapy failed to give relief.

Past History.—The patient had experienced the usual childhood diseases, but he denied having had any injuries or operations other than tooth extractions. He denied having any serious lung diseases but stated that he had had mild asthma, although he was never treated for this condition. There was no history of allergies or of cardiovascular, liver, or kidney diseases. The remainder of the history was noncontributory.

Physical Findings.—The patient was confined to his bed in acute distress because of a hard, localized swelling over the left parotid gland. This swelling extended from the temporal area down to the region of the submaxillary gland. On palpation, the area was painful, dry, red, and warm. The patient had a fetid breath, and although he could open his mouth only about 1 cm., a heavily coated tongue and several heavily stained teeth could be seen. There was no swelling in the pharynx, and the patient had no difficulty swallowing or breathing.

The patient's temperature at the time of his admission to the hospital was 100.5° F., and there was profuse sweating together with an appearance of general malaise. The sinus rhythm was normal; there were no murmurs; and the lungs were clear to percussion and auscultation. The other physical findings were not remarkable.

Admission Orders.—The following were ordered on admission: blood count and differential count; urinalysis; serology; penicillin, 600,000 units twice daily; hot saline mouth

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irrigations; application of hot magnesium sulfate dressings to the face,[1] forced fluids; Demerol hydrochloride, 50 mg. An F-8 plastic nasal feeding tube was used to deliver a 2,400-calorie diet of Waring food mixture supplemented with Sustagen powder. The patient was scheduled for x-ray examination and incision and drainage the next day.

Radiographic Report.—The radiographs gave no evidence of osseous infection or foreign bodies in either the maxilla or the mandible.

Laboratory Findings.—The results of laboratory studies were reported as follows:

Red blood count—3,600,000
White blood count—12,500
Hemoglobin—76 per cent (11.8 grams)
Coagulation time—3½ minutes
Differential: Segmented neutrophils, 67 per cent; nonsegmented neutrophils, 8 per cent; lymphocytes, 21 per cent; monocytes, 3 per cent; basophils, 1 per cent.
Mazzini test—Negative.
Urine—Negative for sugar and acetone.

Fig. 1.

Tentative Diagnosis.—The condition was tentatively diagnosed as (1) left parotid space abscess; or (2) actinomycosis of mandible (left).

Operation.—After using a nasotracheal anesthetic consisting of nitrous oxide and oxygen, Surital sodium, and Anectine chloride, a 1½ inch curved incision was made at the left angle of the mandible. By blunt dissection, an opening was made into the parotid space to permit the escape of about 100 c.c. of foul-smelling pus. No sulfur granules, which would suggest actinomycosis, were noted. A smear and culture were taken and sent to the laboratory for identification. A rubber drain was inserted, the incision was closed with 000 black silk sutures, and a large handkerchief dressing was applied. The patient left the operating room in satisfactory condition with orders to continue the penicillin, nasal feedings, and hot intraoral saline irrigations.

Postoperative Course.—On the evening following the surgical procedure the patient's temperature rose to 102.8° F., but it returned to normal the following morning. Moderate drainage continued for five days and the temperature remained normal. Infrared heat, pre-

ceded by analgesic balm, was applied to the area from a distance of 30 inches for twenty minutes, three times daily. On the sixth day the sutures were removed and a small dressing was applied.

The patient was able to open his mouth 3 cm. on the tenth postoperative day, and he was discharged on the eighteenth day of hospitalization. The rubber drain was removed on the twentieth day, and by the twenty-seventh day healing had taken place and most of the false ankylosis was gone.

The bacteriologic report revealed hemolytic *Staphylococcus albus,* coagulase-negative, with some streptococci. No actinomycetes were observed or cultured.

COMMENT

The most common cause of parotid space infection is septic parotitis. It is unusual for the parotid space to become infected directly by the teeth[2] without involving the fascial planes and spaces. Areas that could be involved from the maxillary teeth include the lip, the canine fossa, the buccal space, the postzygomatic or infratemporal space, the parotid space, the parapharyngeal space, the pterygoid space, the masticator space, the carotid space, the submaxillary space, and the sublingual space.

A detailed knowledge of the anatomic relationships of these structures and space to the teeth is important for a successful incision and drainage. The most common cause of these abscesses is a mixture of staphylococcus (micrococcus) and streptococcus,[3] although a variety of other organisms may be present.

Keefe[4] noted three general periods of pathologic development in inflammatory processes: (1) the initial developmental period, (2) extension into the fascial planes of the face and neck, and (3) serious complications, such as embolism, pyemia, septicemia, and other intracranial complications.

Infections of the face and neck usually fall into one of the following categories[2]:

1. Acute cellulitis
2. Lymphadenitis
3. Abscess formation
4. Phlegmon
5. Chronic cellulitis
6. Chronic abscess
7. Chronic skin fistula
8. Noma
9. Actinomycosis
10. Tuberculosis

Infections must be evaluated carefully, so that the treatment of choice can be used. Frequently, it is necessary to use a combination of surgical intervention and antibiotic therapy to overcome these infections, as in the case just described.

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THE PLACE OF CORTICOSTEROIDS AND ANTIHISTAMINES IN ORAL SURGERY

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IN THE past twelve years, since Hench and associates discovered the potent anti-inflammatory properties of cortisone, this compound and a series of newer adrenocortical steroids have found wide application in virtually every branch of medicine. The literature is replete with reports on the successful use of corticosteroids, particularly in the fields of rheumatology, dermatology, allergy, ophthalmology, and internal medicine. While the anti-inflammatory action of the corticosteroids is of a nonspecific nature and their mechanism of action is not yet fully understood, these studies leave little doubt as to their effectiveness in the suppression of excessive inflammatory reactions.

Nevertheless, in spite of more than a decade of widespread clinical acceptance, the corticosteroids are used relatively little in oral surgery, where edema and resulting pain, together with trismus, cause serious discomfort in over 75 per cent of the patients. The hesitancy in using corticosteroids appears to be due primarily to three suppositions: (1) possible interference with wound healing, (2) danger of masking postsurgical infection, and (3) possible occurrence of serious side effects. In the light of current knowledge, however, these suppositions can no longer be considered valid objections to the *judicious* use of adrenocortical hormones in oral surgery, that is, to their use in the relatively low dosages and for the short periods of time required in the successful management of postoperative edema.

Although some investigators have held the belief that corticoids may retard wound healing,[1] a number of studies on this subject have failed to yield evidence to support this contention.

When Cole and associates[2] injected cortisone subcutaneously into dogs with experimental linear aseptic wounds, they found "no difference in the rate or quality of healing as determined by histologic examination of the wounds," as compared with that seen in control dogs. In another laboratory study, Shafer[3] found no evidence of retardation of healing within ten days after the intra-

Part of this study was presented at the First Conference on Dental Therapeutics, Athens, Georgia, May 11-12, 1960.

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muscular administration of 2.5 mg. of cortisone to rats, following removal of maxillary first molars.

In a clinical study, Traeger[4] administered cortisone to patients with rheumatoid arthritis who required surgery for a variety of conditions. These patients received cortisone "in therapeutically effective doses preoperatively, during the period immediately following surgery and during the postoperative convalescence" and "showed no abnormal deviations from the normal postoperative wound repair." Rukes and associates[5] used intravenous hydrocortisone in more than fifty surgical cases and found "no case in which there was wound dehiscence due to this medication." More recently, Krasner[6] employed a combination of prednisone and chlorprophenpyridamine in the treatment of 485 oral surgery patients and found that this combination did not interfere with wound healing but, on the contrary, accelerated the healing process.

As for the possible danger of masking infections with the administration of corticosteroids, this can be avoided by careful observation of the patient and by administration of appropriate antibiotics when indicated—either prophylactically or when infection is present.

In view of the relatively low dosages employed and the short periods of administration required, steroid side effects are generally not seen in oral surgery. However, a complete history should be taken routinely, so that the use of corticoids may be avoided in patients with such absolute or relative contraindications as active tuberculosis, peptic ulcer, diabetes mellitus, marked osteoporosis, agitated psychotic states, or herpes simplex ophthalmicus. Patients should also be queried as to whether they have recently been on prolonged steroid therapy or whether they are currently taking steroids. In the former case, it may be desirable to administer a higher steroid dosage than that required for reduction of postoperative edema in order to compensate for surgical stress; in the latter case, no additional steroids may be needed for reduction of edema.

The antihistamines constitute another group of compounds which have been used for a number of years in the control of allergic reactions, including those of an inflammatory nature. The rationale for the use of these agents is based upon existing knowledge of their capability for blocking the effect of histamine. The efficacy of antihistamines in oral surgery, as has been the case with the corticosteroids, has been the subject of controversial reports. Keesling and Keats[7] found antihistamines of little value, while both Macchia[8] and Frogley[9] have obtained encouraging results with these compounds.

Highly encouraging results also have been reported with the use of a corticosteroid-antihistamine combination in oral surgery by Stewart[10-12] and by Krasner.[6] In fact, we are indebted to Krasner for the most apt description of the rationale for the use of such combinations:

> The rationale of therapy with the prednisone–chlorprophenpyridamine combination lies in that prednisone, its corticosteroid component, exerts a suppressive effect on the basic anti-inflammatory process by (1) inhibiting migration of inflammatory cells (leucocytes and macrophages), (2) decreasing vascular permeability and (3) reducing exudation; chlorprophenpyridamine, its antihistamine component, (1) blocks the effect of histamine released as a result of tissue trauma and (2) helps control swelling and pain following surgical trauma by reducing tissue distention.

A two-part study was conducted to determine the relative efficacy of corticosteroids and corticosteroid—antihistamine combinations in the reduction of postoperative edema. The first part consisted of a double-blind study comparing a steroid, a steroid–antihistamine combination and a placebo, while in the second part only the effects of the steroid–antihistamine combination were observed.

MATERIALS AND METHODS

Three preparations were used in the double-blind study: one contained 5 mg. of prednisone per tablet; the second consisted of 2.5 mg. of prednisone, 2 mg. of chlorprophenpyridamine, and 75 mg. of ascorbic acid; and the third, containing no active ingredient, served as the placebo for control purposes. The three preparations were identified only as compounds A, B, and C, and neither the patients nor I knew at any time during the study which medication was given. To ensure absolute freedom from bias, a nurse was instructed to give one of these preparations, in turn, to each postsurgical patient prior to his or her leaving the office.

TABLE I. PATIENT MATERIAL*

PROCEDURES	COMPOUND A (PREDNISONE) 14 MALES, 21 FEMALES AGE RANGE: 10-67 YEARS	COMPOUND B (PREDNISONE–CHLORPROPHENPYRIDAMINE) 11 MALES, 22 FEMALES AGE RANGE: 13-63 YEARS	COMPOUND C (PLACEBO) 16 MALES, 21 FEMALES AGE RANGE: 15-60 YEARS
Multiple extractions with or without alveolectomy or maxillary resection†	30	27	29
Single extraction†	3	1	6
Root extraction		1	
Alveoplasty		1	
Apicoectomy		1	
Frenumectomy	1	1	
Caldwell-Luc operation		1	
Exposing of unerupted tooth	1		
Excision of cyst			2
Totals	35	33	37

*One hundred five cases (41 males, 64 females); age range, 10 to 67 years.
†In a large proportion of extraction cases, the patients had impacted teeth.

No effort was made to select patients. The three medications were given to 105 consecutive oral surgery patients from my private practice. The three compounds were alternated, with the first patient receiving compound A, the second compound B, the third compound C, the fourth compound A, and so on.

The 105 patients studied included forty-one males and sixty-four females ranging in age from 10 to 67 years. Thirty-five patients received compound A, thirty-three received compound B, and thirty-seven received compound C. The slightly lower number of cases receiving compounds A and B is explained by the fact that six patients in these two groups failed to return for their postoperative visits and thus had to be eliminated from the study. As Table I shows,

the three groups were made up of patients undergoing substantially similar procedures. The majority had multiple extractions with or without alveolectomy or maxillary resection; a number of other procedures were also included in the study.

In the second part of the study, the prednisone–chlorprophenpyridamine combination was given to fifty-six consecutive oral surgery patients (thirty-three males and twenty-three females, ranging in age from 14 to 63 years). This group included thirty-seven cases of multiple extractions with or without alveolectomy or maxillary resection, eighteen single extractions, and one apicoectomy.

All patients in both studies were given one tablet four times a day for five days and were seen for evaluation on the first or second postoperative day and again on the sixth or seventh postoperative day. Except for codeine or aspirin, which were given where indicated for pain relief, the patients received no other medication.

POST-SURGICAL EDEMA STUDY **Dr. Conrad J. Spilka**

Name _____ **Age** _____ **Sex** _____

Procedure _____ **Date** _____

Compound _____

Other Medications _____

Date	Less than expected edema	Expected degree of edema	More than expected edema

Observations

Fig. 1.—Form used for results in both parts of study.

The form shown in Fig. 1 was used for recording the results in both parts of the study. Patients were evaluated to determine whether the degree of edema was "less than expected," "expected," or "more than expected" with the procedure carried out in each case.

RESULTS

Upon completion of the double-blind study, the pharmaceutical manufacturer who had kindly supplied the active compounds* as well as the placebos furnished us with the code, and the results were evaluated by us.

*The prednisone (Meticorten), the combination of prednisone and chlorprophenpyridamine (Metreton), and the placebos used in this study were supplied through the courtesy of the Medical Services Department, Schering Corporation, Bloomfield, New Jersey.

TABLE II. POSTOPERATIVE EDEMA IN 105 CASES

MEDICATION	DEGREE OF EDEMA		
	LESS THAN EXPECTED	EXPECTED	MORE THAN EXPECTED
Compound A (prednisone) 35 patients	20 (57%)	14 (40%)	1 (3%)
Compound B (prednisone, chlorprophenpyridamine, and ascorbic acid) 33 patients	27 (81.8%)	4 (12.1%)	2 (6.1%)
Compound C (placebo) 37 patients	12 (34.4%)	18 (48.6%)	7 (19%)

Table II shows the results observed with each of the preparations. Of the total of thirty-five patients who received compound A (prednisone, 5 mg.) four times daily for five days, twenty (57 per cent) had less than the expected degree of postoperative edema, fourteen (40 per cent) had the expected degree, and one (3 per cent) had more than the expected degree. In no case was there any detectable interference with wound healing.

In the group of thirty-three patients who received compound B (the combination of 2.5 mg. prednisone and 2 mg. chlorprophenpyridamine) four times daily for a total of five days, the reduction of postoperative edema was dramatic. Twenty-seven patients (81.8 per cent) had less than the degree of edema normally expected following the procedures that were carried out, four (12.1 per cent) had the expected degree of edema, and two (6.1 per cent) had more than the expected degree. It is noteworthy that the majority of these patients reported no pain or minimal pain on the second postoperative day. This may have been due to the exceptionally low degree of edema seen in this group.

The results in the series receiving compound C (the placebo) were rather poor, in that eighteen patients (48.6 per cent) had the expected degree of edema, seven (19 per cent) had more than the expected degree, and twelve (34.4 per cent) had less than the expected degree.

TABLE III. POSTOPERATIVE EDEMA IN GROUP II*
56 CASES (33 MALES, 23 FEMALES)
AGE RANGE—14-63 YEARS

PROCEDURES	DEGREE OF EDEMA		
	LESS THAN EXPECTED	EXPECTED	MORE THAN EXPECTED
37 multiple extractions with and without alveolectomy or maxillary resection	28	8	1
18 single extractions (1 infection, 1 pericarditis)	15	3	
1 apicoectomy		1	
Totals	43 (76.8%)	12 (21.4%)	1 (1.8%)

*Fifty-six cases (33 males, 23 females); age range, 14 to 63 years.

Fig. 2.

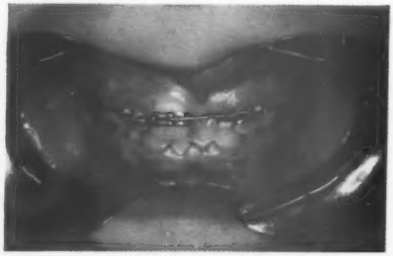

Fig. 6.

Fig. 10.

Fig. 2.—Intraoral photograph prior to excision of tooth.

Fig. 3.—Surgical exposure of unerupted tooth. Considerable trauma is associated with this procedure.

Fig. 6.—Intraoral photograph showing prominent maxillary bone before maxillary resection.

Fig. 8.—Intraoral photograph showing absence of swelling following maxillary resection.

Figs. 10 and 11.—Intraoral photographs before correction of maxillary protrusion.

Following evaluation of the results of the double-blind study, it was decided that the investigation should be extended. In view of the favorable results observed with the prednisone–chlorprophenpyridamine combination, however, it was felt that this medication should not be withheld from patients;

Fig. 4.—Twenty-four hours after surgical exposure of unerupted tooth, there is slight swelling of lip with no discoloration.

Fig. 5. Fig. 7. Fig. 9.

Fig. 5.—Photograph of patient prior to maxillary resection.

Fig. 7.—Photograph of patient twenty-four hours after maxillary resection. Note normal facial appearance.

Fig. 9.—Photograph of patient forty-eight hours after maxillary resection. The facial edema and ecchymoses usually seen are absent.

thus, the study was continued with this combination as the sole agent. As Table III shows, the results obtained confirmed those of the double-blind study; that is, of fifty-six patients, forty-three (76.8 per cent) had less than the expected degree of edema, twelve (21.4 per cent) had the expected degree, and one (1.8 per cent) had more than the expected degree.

Fig. 12.

Fig. 13.

Fig. 12.—Photograph of patient twenty-four hours after surgical correction of maxillary protrusion. Note absence of facial swelling and ecchymoses.

Fig. 13.—Photograph of patient forty-eight hours after surgical correction of maxillary protrusion. Note complete absence of facial swelling and discoloration.

Fig. 14.—Roentgenogram showing odontoma.

Fig. 15.—Photograph of patient twenty-four hours following surgical excision of odontoma. This patient was treated with a placebo. Note extensive edema.

DISCUSSION

Since there are no known means of establishing standards for the exact degree of edema that might be expected with a given surgical procedure in each individual patient, an evaluation of the effect exerted by a drug can be based only upon a comparison with the degree of postoperative edema generally seen with that procedure.

An analysis of the results of the double-blind study shows that 67.6 per cent of the patients receiving a placebo had either the expected or more than the expected degree of edema, while 32.4 per cent had less than the expected degree; of the patients receiving 20 mg. of prednisone a day for five days, 57 per cent had less than the expected degree of postoperative edema, while 43 per cent had the expected or more than the expected degree of edema; of the patients receiving the combination of 10 mg. prednisone and 8 mg. chlorprophenpyridamine daily for five days, 81.8 per cent had less than the expected degree of edema, while only 18.2 per cent had the expected or more than the expected degree.

The results of the study in which the corticosteroid–antihistamine combination was used as the sole agent show that 76.8 per cent had less than the expected degree of edema and 23.2 per cent had the expected or more than the expected degree of edema.

Since the three groups in the double-blind study consisted of a similar proportion of males and females and were made up of patients in similar age ranges who underwent similar surgical procedures, the significant differences in proportion seen among these three groups appear to indicate that, at least in the great majority of cases, the administration of a systemic corticosteroid will substantially reduce postoperative edema following oral surgery and that the addition of an antihistamine to the steroid will not only enhance the latter's effectiveness but will also permit a reduction of its dosage by one-half. Although the risk of side effects is minimal with the relatively small dosages of corticoids and the short periods for which they are given for the control of postoperative edema, the reduction in steroid dosage that may be achieved by the addition of an antihistamine is gratifying, since it reduces even further the side effect potential. It is interesting to note that the results of the study in which only one compound was used closely parallel those seen with the same agent in the double-blind study. In fact, the results obtained with the corticosteroid-antihistamine combination were slightly better in the double-blind study (81.8 per cent had less than the expected degree of edema) than in the study in which I knew that only an active compound was being used (76.8 per cent had less than the expected degree of edema).

Similar results may also be obtained in such surgical procedures as exposure of cuspids, excision of ranulas, removal of hyperplastic tissue, correction of limitation of jaw motion, and correction of mandibular prognathism.

SUMMARY AND CONCLUSIONS

1. A two-part study was carried out to determine the relative usefulness of a corticosteroid–antihistamine combination and of a corticosteroid alone in

the reduction of postsurgical edema. The first part of this investigation consisted of a double-blind study comparing the efficacy of a corticosteroid–antihistamine combination, a steroid, and a placebo. In the second part, only the corticosteroid–antihistamine combination was studied.

2. The judicious use of a corticosteroid–antihistamine combination, such as Metreton, in postsurgical patients will substantially reduce the degree of edema seen following oral surgical procedures.

3. A steroid–antihistamine combination makes it possible to obtain the desired effect with minimal steroid dosages.

4. Pain associated with postoperative edema is minimized, and the need for analgesics is reduced and sometimes eliminated completely.

5. Careful selection of patients is an indicated precautionary measure to avoid administration of a compound containing a corticosteroid to persons for whom these hormones are contraindicated.

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PERIODONTICS

A STENT FOR PERIODONTAL SURGICAL PROCEDURES

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THE recent advances in periodontal therapy with respect to mucogingival surgery and the many periodontal plastic surgical endeavors which have become part and parcel of periodontal therapeutics have imposed on surgical dressings demands which heretofore have been minimal. Because of the intensive procedures carried out today, it is becoming more apparent that the surgical phase of peridontal therapeutics must be consummated not only with as little discomfort to the patient as possible but also with the fewest possible surgical experiences.

Up to the present time, one of the deterrents to the periodontal surgical approach which would facilitate operating on a whole arch at one stage has been dressing retention and functional incapacitation. The postoperative pain and the retardation of healing from loss or loosening of dressings are known only too well. The purpose of this report is to describe a prefabricated surgical stent for the retention of periodontal pack following full-arch surgical procedures. With this stent positive pack retention, mastication on the operative site, and an esthetically satisfactory appearance are possible. The surgical dressing stent is processed in a soft acrylic resin and is contoured to cover the entire buccal and lingual surfaces of the arch for which it is intended.

Impressions of the maxilla and mandible are made, and stone models are poured. The casts are surveyed in light of the surgical procedure to be performed, and the outline is marked. The amount of periodontal pack to be utilized as a covering is estimated, and the outlined area is covered with a wax strip. Duplicate models are then made. A soft acrylic resin material is used, and the standard procedure for fabrication is followed. The advantage of the soft acrylic material is that it can stretch over undercuts and go into place without being forced or severed.

Of importance is the outline of the stent; buccal muscle trim must be carefully planned, since any impingement will result in vestibular fold injury. The palatal coverage is determined by the operative procedure to be performed and by the anatomic features, such as depth of vault and prominence of rugae.

Fig. 1.

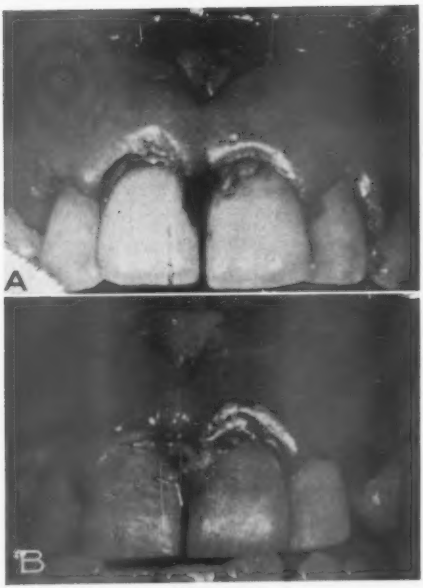

Fig. 2.

The edge of the stent must be smooth and blend with the palatal tissue. In the lingual mandibular region overextension will result in injury, and sufficient clearance must be maintained.

The stent extends as deeply into the embrasure spaces as possible. Any gingival saddle areas in regions of tooth separation or tooth loss are covered completely; the latter aids in the retention of the stent. In instances where the dentition is intact and where there is limited embrasure space, the extension of the papillary gingival portion should be slightly overextended onto the tooth; this will provide sufficient bulk of material in the event an interdental suture should be necessary for the retention of the stent. The material is soft enough for this.

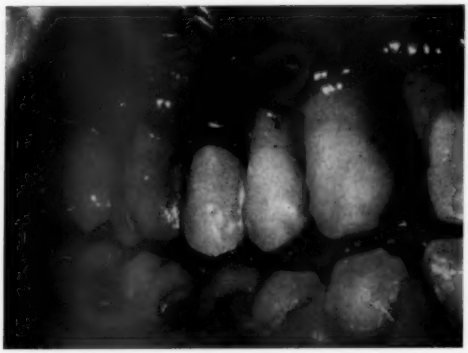

Fig. 3.

Often an anterior tooth or group of teeth may be missing and replaced by means of a removable partial denture. In these cases it is possible to incorporate the missing teeth into the stent to eliminate use of the prosthesis during the healing time and still satisfy the esthetic requirements.

Of advantage also is the possible use of a softer periodontal pack, one which does not need to set completely since it can serve as a dressing and need not serve as its own retention.

Fig. 1 shows complete maxillary and mandibular stents on casts, and Fig. 2, *A* shows the maxillary stent in position immediately after completion of the operative procedure. A suture running labio-palatally was utilized to stabilize the stent. Two weeks later (Fig. 2, *B*) the maxillary stent was still in position, and the suture was still in place. The patient had been able to masticate her usual diet and was satisfied with the esthetic appearance. Upon removal of the stent, it was surprising to observe the relative cleanliness of the operative site. Excessive food retention and debris were minimal. The area was cleansed. Fig. 3 shows the mandibular stent in position and postoperative healing of the maxilla at this time.

ORAL MEDICINE

PRELIMINARY STUDY OF THE EFFECTS OF A STRONTIUM CHLORIDE DENTIFRICE FOR THE CONTROL OF HYPERSENSITIVE TEETH

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THE cause or causes of dental sensitivity have never been fully elucidated, although a number of theories have been advanced in explanation. Colaneri[1] has summarized three possible mechanisms:

1. Odontoblastic processes, being irritable, carry the stimulus (which may arise from changes in the thermal, sugar, or acid environment in contact with the tooth surface) to the nerve plexus of the pulp.
2. Unmyelinated neural fibrils within the dental tubules conduct sensation to the paraodontoblastic nerve plexus.
3. A change in surface tension at the free surface apertures of the dentinal canals causes a disturbance of the hydrostatic equilibrium within the canals which then registers on the subodontoblastic nerve plexus, causing a painful reaction.

Espeland[2] has proposed that hypersensitive dentine is directly responsible for most pulpal sensitivity. Dentine becomes sensitive when its protective enamel covering is lost by abrasion, erosion, or superficial caries. The sensitivity is usually located at the gingival margin where recession has exposed cementum, and its incidence and severity are directly proportional to the amount of organic matter present. Secondary dentine is more resistant to the passage of external stimuli, since it contains fewer accessible dentinal tubules; thus, it is more protective to pulpal tissues. It does not seem likely that the pain in hypersensitive dentine arises from the direct stimulation of nerve endings or nerve fibers in the tubules. Application of agents that can stimulate nerve-ending response in other parts of the body does not produce dentinal pain.[3]

The widely accepted explanation that hypersensitivity arises from the transmittance of peripheral stimuli, originating on the tooth surface, through

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the fluid or semifluid content of the dentinal tubules to the pulp surface was questioned, however, by Kramer.[4] In a histologic study of 152 teeth filled with unlined resins, Kramer found no essential differences in pain between teeth in which odontoblasts or odontoblastic nuclei had been aspirated into dentinal tubules opened during cavity preparation and those in which aspiration had not occurred. Since violent disturbances must have accompanied the aspiration, he believed that the theory was improbable.

Regardless of the mechanism giving rise to painfully sensitive dentine and cementum, there is no question that the condition is annoying or troublesome to the patient who is afflicted and to the dentist who is expected to provide relief. In some patients, pain can be so severe as to constitute a physical and emotional problem. Many patients avoid mastication of tough foods because of the sharp tactile pain that occurs on contact with the tooth surface. Others are no longer able to enjoy heated or chilled liquids or foods because of the painful sensations which they evoke. The effects of acid and high-carbohydrate foods in stimulating painful sensations are also well known.

Many methods have been proposed for treating dentine and cementum sensitivity; most practitioners have a favorite office approach. Among the agents in common use are formaldehyde solution (10 per cent to 40 per cent), hot olive or mineral oil, silver nitrate (10 per cent to 35 per cent), ammoniacal silver nitrate solution N.F., zinc chloride (10 per cent), sodium fluoride-kaolin-glycerin paste,[5] pure glycerin,[1] sodium silicofluoride solution,[6] sodium potassium carbonate paste,[7] creosote,[8] liquefied phenol,[8] benzyl alcohol,[8] eugenol,[8] absolute alcohol,[9] trichloroacetic acid,[9] and a mixture of caustic potash and phenol in glycerin (Robinson's remedy).[9]

One explanation of the mechanism of effect of some of these agents is that they operate by destroying the odontoblastic fibrils, thereby preventing sensory impulses from originating or from reaching the dental pulp tissue.[8] Another possibility is that agents which can bring about dehydration or blocking of the perifibrillar space by occlusion or by secondary dentine formation will relieve the condition.[10]

Major disadvantages of many of these therapies are their restriction to office use, their frequently inconsistent effectiveness, their potential for systemic toxicity, or, in some cases, their local irritant or discoloring effects. Silver nitrate, for example, is reasonably effective but its use on exposed surfaces is contraindicated because of its discoloring tendencies.[9]

There is a long-felt need for a nontoxic, nonirritating, and nondiscoloring agent which could be used with impunity by the patient under a normal home-care regime.

Fitzgerald[11] has reported on the use of a desensitizing dentifrice among ninety-two patients with an average duration of dentine sensitivity of 4.6 years. "Complete" or "good" relief was found in 72 per cent of the cases. One patient reacted to the product with a reddening and irritation at the gingival margin.

Abel[12] studied the same dentifrice, which was stated to contain calcium carbonate, sodium bicarbonate, sodium chloride, sodium and potassium sulfates,

and a small percentage of formalin, and found complete to moderate relief in 77 per cent of patients using the product. Toto, Staffileno, and Gargiulo[13] also reported on the same dentifrice, which they said contained 1.4 per cent formalin, and found that forty of forty-one subjects using it were free of complaints of sensitivity after three weeks, while patients using an identical formulation without the formaldehyde experienced a diminution of sensitivity which, however, did not completely disappear even after five weeks.

On the basis of the study by Toto and his colleagues,[13] it appears that formaldehyde in the dentifrice studied is primarily responsible for the favorable results in the three reported clinical studies of the product and that the other constituents play a secondary role. Dobbs and Prinz[8] have stated that formaldehyde is an irritant to the mucous membrane, even in dilute solutions; formalin dermatitis is believed to arise from a lessened resistance of the patient to continuous exposure to the drug.

My interest in the problem of hypersensitive teeth and their possible treatment was restimulated by a recent report in the Polish literature.[14] An aqueous solution of strontium chloride was reported to be effective in relieving sensitivity by virtue of a biocolloidal binding and blocking action with or in the organic matrix of tooth structure. Teeth treated with the solution were reported to become harder, smoother, and more lustrous.

The physical and physiologic characteristics of strontium chloride seemed to be rational for the intended purpose. It is a neutral salt of excellent water solubility with low systemic toxicity, being somewhat less toxic than calcium chloride.[15] Strontium compounds generally behave chemically like their calcium analogues, and in some physiologic processes they can substitute, in part, for calcium.[16] Strontium compounds have been found to deposit in human bone[16] and to be useful for remineralization of the osteoporotic skeleton.[17]

Hodge, Gavett, and Thomas[18] have demonstrated that strontium chloride deposits significant amounts of strontium by adsorption on powdered bone, dentine, and enamel. The uptake of strontium by bone and dentine was markedly greater than fluoride at 40° C., although enamel adsorbed more fluoride. Buonocore and Bibby[19] have shown a slight reduction in the solubility of powdered enamel following exposure to strontium ion, which may be indicative of a partial replacement of calcium in tooth apatite.

Strontium administered by injection in high concentrations cannot replace calcium during matrix formation in dentine and enamel, since tooth structure develops abnormally,[20] but low systemic concentrations have little effect on normal enamel formation.[21]

After I became interested in strontium chloride as a possible desensitizer, I first used it in a 25 per cent liquid solution which was applied topically to cementum that had been exposed by gingivectomy. It was effective, but for only a short period; reapplication was necessary. A dentifrice* in which the agent was employed in a 10 per cent concentration was offered for clinical appraisal. The product, at that time identified by code number

*Sensodyne, manufactured by the Block Drug Company, Inc., Jersey City, New Jersey.

only in plain white tubes, was a conventional-looking toothpaste with seemingly adequate foaming and polishing action and a distinctive, although not unpleasant, flavor. According to the research laboratory of the manufacturer, the product was formulated in keeping with the reactive chemical nature of the active ingredient; calcium- and magnesium-based polishing agents were avoided because of possible cationic replacement by strontium; both soluble and insoluble phosphates and carbonates were similarly contraindicated to avoid reaction with and subsequent precipitation of water-insoluble strontium-anion derivatives. Laboratory studies were said to demonstrate the almost complete availability of strontium ion in aqueous extracts of the dentifrice, which I believed to be an important consideration for the objective evaluation of whatever effects solutions of strontium compounds might have. The formulation was presented as containing the following ingredients:

Strontium chloride · $6H_2O$	10.0 per cent
Deionized water	36.8 per cent
Polyols	24.0 per cent
Water-soluble gums	1.8 per cent
Anionic detergent	1.7 per cent
Nonionic detergent	0.5 per cent
Diatomaceous silica	23.0 per cent
Flavor and saccharin	1.35 per cent
Inert coloring agents	0.75 per cent
Preservative	0.10 per cent
	100.00 per cent

Patients undergoing periodontal treatment are particularly suited for the evaluation of desensitizing agents. The exposure of cementum following surgical procedures, such as gingivectomies and osteotomies, frequently aggravates an already serious condition and often results in thermal, ionic, or tactile sensitivity that may range from annoying to exquisite pain to the patient.

It has been my practice for several years to prescribe either flavored or unflavored mouthwash solutions of sodium silicofluoride[6] for home use. Results have been quite favorable, but there were some limitations to continued use by the patient. The mouthwash is not commercially available, and the patient could not always be relied upon to have prescriptions refilled or to obtain additional supplies from my office. There was also the additional problem of potential toxicity for young children at home who might accidentally ingest the entire contents of a bottle.

It has been my experience that almost none of the office treatments employed for the relief of hypersensitive teeth is permanent, thus emphasizing the need for a home adjunct. Periods of relief following office application of desensitizing agents range from a few days to a few months or more, but there is frequently a recurrence which requires repeated treatment.

The strontium ion dentifrice has been dispensed to about 100 patients diagnosed as having hypersensitive dentine, and records of effectiveness are

TABLE I. TREATMENT OF HYPERSENSITIVE CEMENTUM WITH STRONTIUM CHLORIDE DENTIFRICE

RESULT OF TREATMENT	NO. OF PATIENTS	PER CENT
Complete	54	66.6
Good	7	8.6
Fair	10	12.4
Little or none	10	12.4
Totals	81	100.0

available on eighty-one of them. All patients given the toothpaste were instructed in adequate home care. Results of the use of this dentifrice are shown in Table I.

It can be seen from Table I that use of the strontium ion dentifrice induced good to complete relief in 75 per cent of the patients using the product, while some effectiveness was noted in 87 per cent of the patients. Relief was generally noted within a week or two after first use, but in some patients it was three to four weeks before a noticeable response was achieved. Cases which did not respond within a two- to three-month period were considered refractory.

In nearly all cases in which accurate period records were possible in spite of the sporadic nature of patient recall, it was noted that relief was frequently progressive; that is, the typical patient who experienced some relief within a few days found that diminution of sensitivity continued to build up with concientious use of the dentifrice. Consequently, the records of the general progress of many patients show a reclassification of effectiveness from "fair" to "good" or from "good" to "complete" on repeated visits.

Evaluation of effectiveness was made by subjective evaluation by the patient on verbal interrogation prior to any experimental procedures and again after the use of two techniques that are generally painful to sensitive tooth structure. In the first of these techniques the point of an explorer was pulled lightly over affected areas, and in the second the patient rinsed his mouth with cool water. In many instances it was not necessary to obtain the patient's subjective evaluation, since the absence of the customary wince or facial grimace following the procedures indicated that pain had been obtunded, although excellent agreement has been found between the patients' comments and my own objective observations.

There has been no evidence of tissue sensitivity or reaction in any of my patients after twice-daily use of the dentifrice for periods of up to one year. The reaction of patients to the dentifrice as a dentifrice has been generally good. A few isolated comments on flavor, which were probably influenced by the salty and slightly bitter character of the strontium chloride, have been noted, but none of the patients has refused to use the product and most have commented on its refreshing character as well as on its therapeutic effectiveness.

As for the cleaning and polishing characteristics my observations after prolonged use have shown that in these respects this dentifrice is typical of most conventional dentifrice formulations. Patients with a tendency to accrue calculus accretions continue to do so at about the same rate, whereas those with adequately self-cleansing mouths appear equally unaffected.

DISCUSSION

It has been pointed out that secondary dentine formation reduces the passage of external stimuli.[2] Calcium hydroxide, used as a liner under silicate cement, will usually reduce or eliminate sensitivity in the dentine of the prepared cavity.[22] It is not definitely known by what physiologic mechanism external stimuli are transmitted to the pulp; empirically, however, many agents which can block the transmission of stimuli seem to do so either by destroying odontoblastic fibrils or by forming a mechanical barrier within or at the ends of the dentinal tubules. Strontium chloride has been shown to have a strong adsorptive capacity for calcified tissues, especially for calcified tissue with a high organic content.[18] It does not seem unlikely that the mechanism of effectiveness in reducing or eliminating dentine sensitivity may arise by virtue of its adsorption on the organic matrix of dentine and especially on the termini of or within the dentinal tubules, with corresponding blocking of impulses to neural centers. It is also conceivable that it may serve as a bridge for stimulating secondary dentine calcification within the odontoblastic processes, which could further serve to block sensory impulse. This possible explanation has some support in the findings of Shorr and Carter[23] that calcium remineralization of the skeleton was considerably higher when strontium and calcium were administered together than when calcium alone was used, no matter how high the calcium intake. Obviously, some of these hypotheses are conjectural, however, and require the evidence of further in vitro and in vivo exploration.

The demonstrated effectiveness of strontium chloride in a neutral, compatible dentifrice base in controlling dental hypersensitivity seems to be an important advance in dental therapeutics. For the first time, it appears possible that a nontoxic, nonirritating, and nonallergenic chemical agent can be used for the daily treatment of a most common dental pain problem. So far as I know, there are no limitations or contraindications to the use of strontium chloride or of a neutral dentifrice containing this agent.

SUMMARY

A 10 per cent concentration of strontium chloride, in a neutral, compatible dentifrice base which releases strontium ions when in contact with water, has been demonstrated to be useful in controlling the pain arising from hypersensitive cementum in about 75 per cent of the cases studied.

I know of no contraindications to the use of strontium chloride for the intended purpose, since as it is used it is apparently nonirritating, nonallergenic, and nontoxic. Its incorporation into a dentifrice seems to make possible, for the first time, a rational, safe, effective therapy that can be used in a daily regimen by the patient suffering from hypersensitive cementum. It is suggested that the results obtained in this study may provide an incentive for additional histologic and laboratory studies on the mechanism of action of water-soluble strontium as well as further clinical evaluations of these compounds and of dentifrices containing them.

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PYOSTOMATITIS VEGETANS

Report of a Case

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In 1898 Hallopean[1-3] described a skin lesion in adults which was characterized by successive crops of pustules or vesicopustules. This lesion occurred chiefly about the genitals, but it was also seen in the hypogastric and lumbar regions, in the axillae, on the cheeks, on the chin, and in some instances in small numbers upon the mucous membranes of the lips and tongue. This pustular eruption developed into an elevated, more or less deeply pigmented, vegetating plaque covered with crusts. There appeared to be no definite cause for the disease, but an eosinophilia was usually present. This condition was later described in infants by Wende and DeGroat.[5]

Fordyce and Gottheil[6] reported the condition to be analogous to dermatitis herpetiformis in its histologic and hematologic characteristics (eosinophilia). In the sites of predilection—the mouth, genitals, and lower extremities—it is very much like pemphigus vegetans; however, its relatively benign course is in striking contrast to the latter disease.

In 1949 McCarthy[9] reported three cases involving primarily the oral cavity and suggested the name "pyostomatitis vegetans" for this condition. In two of his cases no lesions appeared elsewhere. He described the primary oral lesions as minute miliary abscesses of uniform size on a slightly raised dark red inflammatory base. In his patients the process spread within a few weeks to involve the entire oral cavity. As the condition developed chronically, the buccal mucosa began to proliferate and was thrown into folds, assuming a verrucous appearance. The lesions were not painful. Local treatment had no effect on McCarthy's patients, but following prolonged rest and liver and iron injections, two of his patients were reported cured.

Becker and Coventry[10] reported one case of severe recurrent pyoderma with oral manifestations. The condition appeared subsequent to an attack of meningitis, as it did in one of the cases reported by McCarthy. Splenectomy helped but failed to prevent subsequent leukopenia and recurrent infection.

CASE REPORT

A 61-year-old white man was admitted to the hospital with metastatic adenocarcinoma in the supraclavicular area, plus bone metastases in the right shoulder girdle and the right

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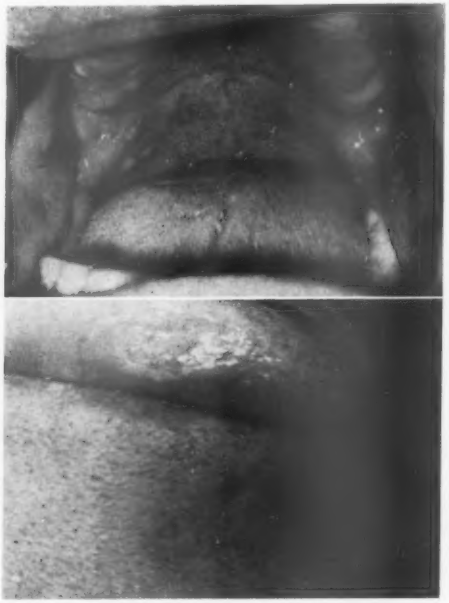
iliac crest. Lung lesions were thought to be probably metastatic or primary and metastatic. White membranous patches were noted on both tonsils, especially the left one, with some surrounding erythema. The membranous lesions also involved the posterior pharyngeal wall, uvula, and anterior tip of the tongue. A biopsy of the tongue was performed at this time, and the patient was discharged on the eighth hospital day to be followed in the tumor clinic.

A.



B.

Fig. 1.

Two weeks later, when the patient was seen in the tumor clinic, there were discrete, thick, plaquelike membranous lesions on the cheek, the palate, the tongue, the posterior ridges of the mandible, and the pharynx (Fig. 1, *A*). Each lesion was circumscribed, presenting a tiny shallow crater with no particular surrounding erythema. The cratered, whitish papule was easily removed, leaving a very shallow, slightly bleeding ulcer which was not especially painful. The lesions were not characteristic of herpetic stomatitis. When the patient was seen two weeks later, a great many of the oral lesions had disappeared. A few remained on the left anterior tonsillar pillar, and there was one on the right lateral margin of the tongue (Fig. 1, *B*). One week later the lesions that remained were smaller, and the following week there were present on each lateral margin of the tongue two small, flattened, whitish areas consistent with the beginning slough of vesicular epithelium. There was no pain or bleeding.

When the patient was seen two weeks later, almost two months after the initial observation, there was a slight increase in the number of lesions, particularly a large, irregularly shaped lesion on the labial gingiva and mucobuccal fold which extended from the area of the left lateral incisor to the area of the right lateral incisor. Several of the smaller lesions could be rubbed off easily, but the new ones were firmly adherent.

Six weeks later all the mouth lesions had disappeared, but when the patient was seen almost a month after that there were two whitish papules on the soft palate near the midline

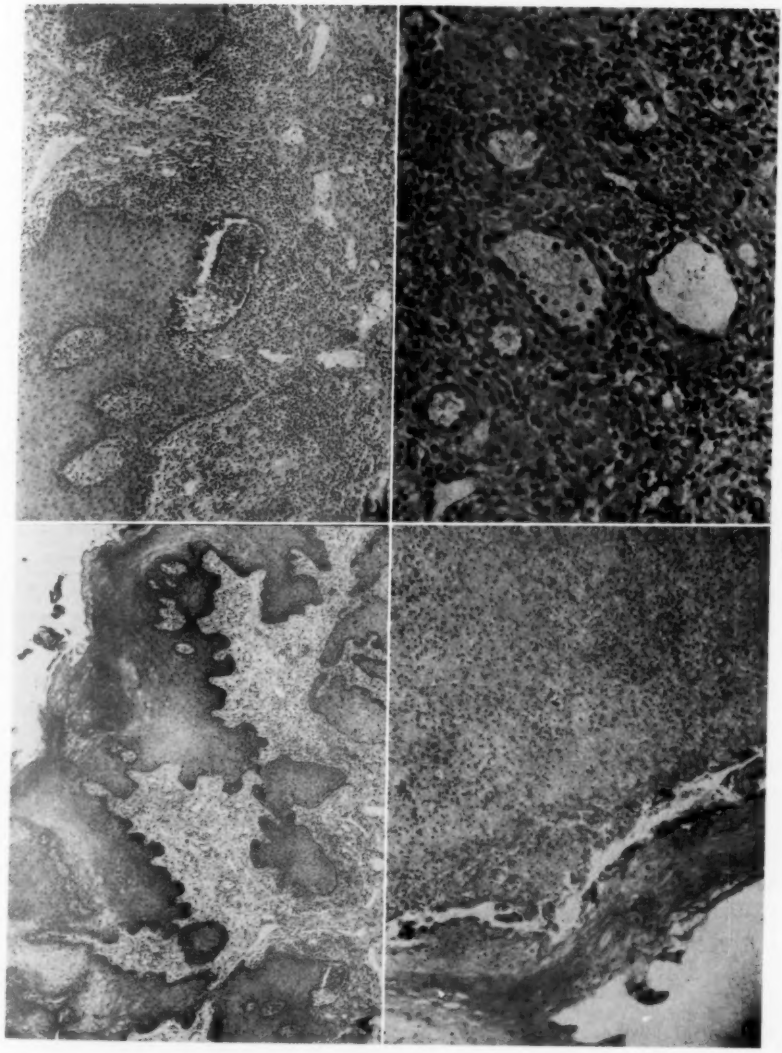


Fig. 2.

and one on the uvula. These lesions could also be wiped off easily, leaving a denuded hemorrhagic spot. When the patient was seen after another two weeks, there were a few small areas on the tongue and soft palate. A short time afterward the patient died of metastatic carcinoma in another hospital.

Examination of Tissue Specimen.—The specimen was a square piece of tongue tissue measuring 1.0 by 1.0 by 0.3 cm. The epithelial surface showed a thick parakeratotic and keratotic layer (Fig. 2, *A*). There was a marked acanthosis with a proliferation of the rete pegs into the underlying corium. The stratum germinativum showed a normal cell morphology, however. The epithelium was intact throughout the specimen, except in one area where an ulcerative surface was present (Fig. 2, *B*). Several minute unruptured abscesses were present within the epithelium. The corium showed an increased number of small blood vessels and a great many lymphocytes, plasma cells, and hitiocytes (Fig. 2, *C*). The predominating cell, however, was the eosinophil (Fig. 2, *D*). The connective tissue fibers had undergone degeneration. The cellular infiltrate was most marked in the subepithelial layer of the corium. The over-all picture was that of chronic granulomatous tissue.

DISCUSSION

I believe that the case described fits the description of McCarthy's three cases of pyostomatitis vegetans. Unfortunately, it was not possible to observe the patient for a long enough period of time to see the final course of the lesions. During the time that the patient was under observation, there seemed to be remissions and exacerbations of the lesions, which was not true of the cases described by McCarthy. Local treatment proved ineffective in our patient.

SUMMARY

The case history of a patient with pyostomatitis vegetans has been presented. The condition was characterized in its early stage by painless minute miliary abscesses; later, the lesions became verrucous in character. Microscopically, there was a marked acanthosis and keratosis. Microscopic abscesses were present in the epithelium. There was a dense cellular infiltration of the tunica propria, with the eosinophil predominating.

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601 Portland Medical Center.

A STUDY OF SOME PERIAPICAL ROENTGENOLUCENCIES AND THEIR SIGNIFICANCE*

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It has been demonstrated that some agent capable of causing periapical irritation is generated during pulpal infection. The agent reaches its site of action by passing through the apical foramen.[1] When the irritant has been eliminated, healing will occur.[2]

The reaction of tissue to irritants—either bacterial, physical, or chemical—has the same basic response, whether the reaction be within the tooth or not. These irritants, when introduced into the tissue, may elicit a response commensurate with their destructive potency. The response may produce a slight tissue reaction if the stimulus is mild. When the stimulus increases, the response reaches a stage where the resistance of the tissue cannot localize and contain the action. This will result in tissue breakdown and in the formation of pus.

The reaction of the host to the irritant is determined by the contact of the organism with the tissue, the amount of toxic material emanating from the nidus, and the host's susceptibility to these irritants.[3] It is the delicate balance of action and counteraction that usually maintains or attempts to maintain the status quo.

This study was undertaken to determine the incidence of positive cultures obtained from periapical tissues. Granulation tissue is healing tissue[4]; therefore, its removal only delays repair. Surgical removal of involved periapical tissue is logical only if periapical tissue is infected.

TYPE OF STUDY AND METHOD

The scope of this study was limited to determining whether or not apical granulation tissue harbors bacteria. No identification of the types of microorganisms was planned.

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As noted in the résumé of the literature, many investigators have developed experimental techniques designed to determine the bacterial content of both the root canal and the periapical granulation tissue. Cramer and Reith,[5] Boyle,[6] and Hardnt[7] used extracted teeth. Bulleid[8] used three types of specimens in his bacteriologic study of periapical infections. These included (1) extracted teeth, (2) specimens obtained by cutting through alveolar plate, and (3) apical granuloma attached to the tooth after extraction. Grossman and Prinz[9] used a trochar and cannula and drilled through the alveolar plate for the culture. Burket,[10] who used teeth from autopsy specimens, also cut through the alveolar plate for his culture after sterilizing the superficial tissue.

In a recent study by Hedman,[11] the teeth were studied in situ. Using cannulas and stylets, Hedman took cultures of the residual periapical areas. The initial procedure was that of culturing the content of the canal. After débridement by oxidative lavage, the cannula was inserted to the apex. Cultures of the apical granulomatous area were obtained by passing a stylet through the tooth apex.

All of these studies were made for bacteriologic evaluation. Although the procedures were varied, the basic bacteriologic techniques were similar as far as procedure and culture media were concerned. The conclusions drawn show diffuse and inconsistent results.

Most early bacteriologic studies of the apical areas were carried out following extractions. Although there were various attempts at aseptic techniques, it is nevertheless possible that the positive results were due to contaminants. Hardnt,[12] using extracted teeth, found that all solid granulomas are sterile. He studied the granulomas histobacteriologically and found that bacteria were never in or among the cells of the granuloma. Any bacteria found on the outer surface of the capsule were contaminants from the extraction process. Fish and MacLean,[13] using a similar procedure, concurred and claimed that living tissues are invariably sterile. Streptococci, when found, are usually confined to acute or chronic abscess areas. Osgood,[14] in a roentgenographic evaluation, found that granulomas occur in young adults whose resistance is high. Chronic abscesses tend to occur in patients with lowered resistance. Kronfeld,[15] in a histobacteriologic study of pulpless teeth in situ, always found bacteria in the canal, but granulation tissue attached to the apices of the teeth was often free of microorganisms. He determined that granulomas and cysts are sterile in a large percentage of the cases. He stated that granulomas destroy bacteria, for this tissue is "one of healing."

Conversely, Cramer and Reith,[16] reporting a study of extracted teeth, stated that "(1) granulomas are invariably infected, (2) apical areas about x-ray positive teeth are probably infected, (3) teeth with slight periapical change are infected to a lesser degree than those with more pronounced pathology, (4) teeth definitely x-ray negative are infected only occasionally." Gilmer,[17] who studied extracted teeth from forty cases, found aerobic as well as anaerobic organisms in the periapical tissues. In addition to extracted teeth, Burket[10] studied teeth at autopsy by cutting through the alveolar plate. He stated that granulomas

were 49 per cent positive for bacteria. In a further evaluation by means of x-ray, he found a decrease in the percentage of pure cultures as the size of the periapical roentgenolucent areas increased. As the size of the periapical lesion increased, mixed cultures were found more frequently. Haden[18] cultured 1,307 vital and nonvital extracted teeth. In comparing them with their Roentgenograms, he found the incidence of positive cultures about the same in x-ray-negative and x-ray-positive teeth. Rickert[19] cultured through the apical bone and found that the granulomas were bacteriologically positive.

In a survey of the foregoing techniques one feature seems to repeat itself: each procedure was open to contaminants. Burket[10] could not show a completely sterile technique when he used extracted teeth for his experiments. Grossman,[20] who used a trochar and cannula, and Bulleid,[8] who cut through the alveolar plate, used procedures that were subject to contamination. Hedman[11] used a procedure that eliminated the extra dental potential for contamination. His procedure was to clean the canal by chemical débridement, then to pass a cannula with a wax plug over its orifice to the apex, and next to warm the stylet and push it through into the apical pathologic area. Of the eighty-two cases in his study, sixty-three had microbial growth in the canals; fifty-six of this, latter group also had growth in the periapical area. Seven that showed positive cultures in the canal had negative growth in the periapical area. After negative cultures in the canals were obtained, infection in the periapical tissue was resolved.

The purpose of this study was to ascertain a true bacteriologic picture of the apical area within technical limits. It was thought that if the canals were mechanically cleaned, the possibility of pushing any of the contaminants ahead of the cannula into the periapical tissue would be practically eliminated.

In this study there were no limitations as to specific age group, size of the apical area, acute or chronic symptoms, or other related factors. The essential requirement was that there be roentgenographic evidence of a periapical area. Types of specimens were not limited; all were routine cases accepted by the clinic for treatment. Neither exposure of the canals to the mouth fluids or a history of healed fistulous openings had any bearing on their acceptance. Because of their accessibility, most of the teeth studied were anterior teeth. A 26 gauge needle was used as a cannula and therefore required a canal reamed to the size of a No. 6 reamer in order to permit passage to the apex. The stylet had a 45 degree angle bend, so that when it was pushed beyond the cannula it would spring out to its predetermined angle. In this way, it would be in position to sweep around the apical area, and to collect samples for culturing. The cannulas and the encased stylets were autoclaved and stored in a Petri dish.

The technical procedure followed accepted methods of root canal therapy, including the placing of a rubber dam and painting of the area with Metaphen followed by Zephiran chloride. The tooth was opened by means of a sterile bur. A paper point was inserted at the orifice and cultured in an enriched medium. The length of the tooth was determined by means of a No. 1 file, making sure that it stayed at least 1 mm. short of the apex. Reaming and filing were then done

interspersed with sodium hypochlorite flushing of the canal. This mechanical débridement was done with the utmost care so that possible contamination through the apex would be held to a minimum. When the canals were completely débrided, at least to the size of a No. 6 reamer, a second paper point was inserted just to the apex and cultured. The third culture was then taken. The autoclaved cannula was inserted by means of sterile forceps to the apex and the stylet was pushed through until the angle was completely free of the cannula's confines. The stylet at the crown orifice was bent at a right angle and then turned 360 degrees several times in an attempt to contact all parts of the periapical area. The stylet was then withdrawn so that the part contaminated by the operator could be cut off with sterile shears. The rest of the stylet was withdrawn with sterile forceps and dropped into the culture tube. The tubes were incubated at 37° C. for at least seventy-two hours before being read.

To determine the feasibility of this technique and eliminate any potential discrepancies that might cloud the results, the following preliminary tests were run. The stylet was roughened with a stone, so that when the wire was withdrawn the loss of microbes against the sides of the cannula would be minimal. The cannulas and stylets were cultured to make certain that they were not oligodynamic. They were cultured on nutrient agar and blood agar plates with debris from positive-cultured canals. As a further test of the feasibility of using this stylet for culturing, cannulas were inserted into the positive-cultured canals. With the same technique as that used for the periapical procedure, the stylet was pushed through to free the angle and then turned 360 degrees several times to contact all the walls. The stylet was then withdrawn in like manner and cultured. All cultures were positive.

TABLE I. POSITIVE CULTURES IN DIFFERENT PARTS OF TEETH WHICH HAD PERIAPICAL ROENTGENOLUCENT AREAS

CASE NO.	CULTURES			CASE NO.	CULTURES			CASE NO.	CULTURES		
	A	B	C		A	B	C		A	B	C
1	−	−	+	22	+	+	−	43	+	−	−
2	+	−	−	23	+	−	−	44	+	+	−
3	+	−	−	24	+	−	−	45	+	+	+
4	+	+	−	25	+	+	−	46	+	−	−
5	+	−	+	26	+	+	−	47	+	−	−
6	+	+	−	27	+	−	−	48	+	−	−
7	+	−	−	28	+	−	−	49	+	+	−
8	+	−	−	29	+	−	−	50	+	−	−
9	+	−	−	30	+	−	−	51	+	+	−
10	+	+	−	31	+	−	−	52	+	−	−
11	+	−	−	32	+	−	−	53	+	+	−
12	+	−	−	33	+	−	−	54	+	+	−
13	+	−	−	34	+	−	−	55	+	+	−
14	+	−	−	35	+	+	−	56	+	−	−
15	+	+	−	36	+	+	−	57	+	−	−
16	+	−	−	37	+	−	−	58	+	−	−
17	+	−	−	38	+	−	−	59	+	−	−
18	+	−	−	39	+	−	−	60	+	+	−
19	+	+	−	40	+	−	−	61	+	−	−
20	+	+	−	41	+	+	−	62	+	−	−
21	+	−	−	42	+	−	−	63	+	−	−

A—Prereaming culture.
B—Postreaming culture.
C—Periapical culture.

The culture media were Difco enriched beef-heart infusion broth, Tryptocase soy broth and 0.1 per cent agar. The Difco broth was used for the first thirty-five cases, and the remaining cases were cultured in the Tryptocase soy broth and the 0.1 per cent agar. The cases that showed the third culture tube positive were plated on blood agar. It was found, coincidentally, that the microorganisms consisted solely of staphylococci and streptococci.

RESULTS

Sixty-three cases were studied. All of these indicated definite roentgenographic periapical areas. There were no limitations as to sex, age, or clinical symptomatology. Several patients had histories of previous exacerbations.

Table I presents the findings. The cultures that were initially positive are shown, followed by the postreaming cultures that were positive and finally by the periapical cultures.

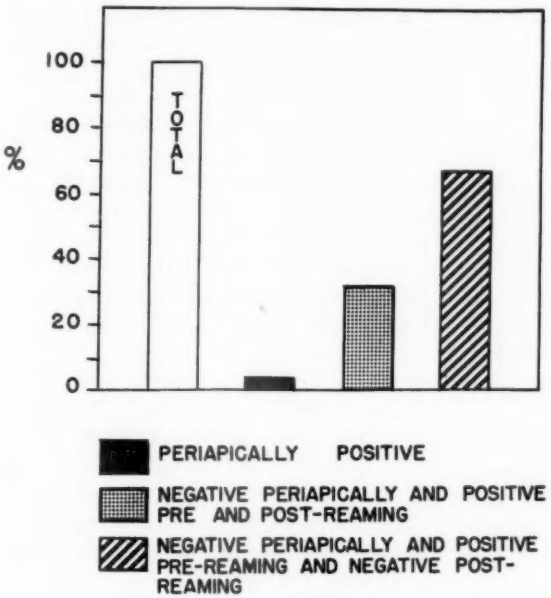

Fig. 1.

Of the sixty-three cases studied, three showed positive cultures for the periapical tissue. Sixty cases were negative. One of the three positive cases had negative pre- and postreaming cultures, the second case had positive pre- and postreaming cultures, and the third case was positive throughout. Of the remaining sixty cases forty-one were initially positive but had negative postreaming and periapical cultures. Nineteen were positive both before and after reaming and negative periapically; there is a statistically significant difference (chi-square = 35.3) between the incidence of postreaming positive cultures and the incidence of apical positive cultures.

A summary of cases, as shown in Fig. 1, is as follows:

1. Of the sixty-three cases, 5 per cent were periapically positive.
2. 30 per cent of the cases were positive both pre- and postreaming and negative periapically.
3. 65 per cent of the cases were positive the prereaming and negative both postreaming and periapically.

The patients in this study ranged in age from 11 to 72 years. A tabulation of results by sex showed that 25 per cent of the male patients had two positive cultures and 75 per cent had only one. The figures for female patients were 36 and 64 per cent respectively. A chi-square test supported the null hypothesis (chi-square = 0.385). The same type of comparison was made for patients older and younger than 23 years, with no indication of a difference in distribution attributable to age (chi-square = 0.429).

Fifty-one patients had necrotic pulps and were asymptomatic clinically. Thirty-six of these showed positive prereaming cultures. The postreaming and periapical cultures were negative. In the remaining fifteen cases cultures were positive both before and after reaming and were periapically negative. The potency of the bacterial and toxic reaction is evidenced by the high incidence of periapical alterations as shown roentgenographically. In spite of bacterial growth in the canal, the absence of positive periapical cultures tends to demonstrate the resistance of the altered apical tissue to invasion by bacteria. In the course of the study, four cases were found in which there were histories of healed fistulas. Subperiosteal swelling was evident in one case, although the patient did not complain of any discomfort. Both the pre- and postreaming cultures were positive. A negative culture was obtained from the periapical area, indicating probably a toxic reaction around the apex rather than bacterial invasion.

Three cases showed clinical evidence of pericementitis. Although all the periapical cultures were negative, there may have been an increased mechanical or toxic reaction of the periodontium in these patients. Two of the cases showed postreaming positive readings.

Four cases were asymptomatic clinically, even though a purulent exudate was observed when the canal was opened. In, all four cases cultures were positive bacteriologically before reaming; two were positive after reaming, and the other two were negative after reaming. The periapical readings were all negative. The fact that the initial cultures were positive indicates the presence of viable bacteria.

DISCUSSION

The important finding originating from this work is the low incidence of positive cultures obtainable from periapical lesions. This result serves to alter our concepts in several ways. Invasion of the periapical tissue by microorganisms from the tooth canal is not an easy process; this invasion does not occur, or it does not occur rapidly enough for the microorganisms to gain a foothold. The

findings also show that roentgenographic evidence of periapical lesions does not necessarily mean that microorganisms are present in the lesions. Furthermore, since there are few cases of infection of periapical tissue, roentgenolucent areas of periapical tissue cannot be interpreted as predisposing to blood-borne microorganisms.

This conclusion is based on the assumption that the majority of negative cultures are attributable to the absence of organisms. It is recognized that a negative culture is not always an assurance that all bacteria have been eliminated. Sometimes it may indicate only that the number of bacteria present are below the threshold of sensitivity.

Some mention should be made regarding the technique of employing sodium hypochlorite solution (Zonite) as an oxidizing agent and lubricant while reaming a canal. It is theoretically possible that this treatment might have exerted a bactericidal effect which would serve to reduce the incidence of positive cultures taken from the granuloma. The treatment occurred between the first and second cultures taken. I believe that bactericidal action by the sodium hypochlorite is unlikely, since the treatment failed to produce a sterile canal in nineteen cases; there were nineteen cases in which both pre- and postreaming samples were positive. It seems highly unlikely that a treatment which would permit organisms to remain in the canal in 30 per cent of the cases could possibly diffuse into the periapical area with a resulting bactericidal action. Furthermore the final reaming was done without a lubricant; this technique would severely limit the quantity of solution that could be forced through the apical foramen.

An interpretation of the findings can be developed which is consistent with accepted knowledge in the field of pathology. This mechanism probably explains why spontaneous resolution in the periapical area is nonexistent. The lack of tissue fluid interchange in the canal leads to stasis, with subsequent breakdown of the fluid proteins. These breakdown products may serve as a medium for the bacteria present, and only the addition of the necessary adjunctives to their growth permits them to flourish. As the pulp tissue dies, the interchange of the tissue fluids is reduced until there is a complete stasis. The tissue then becomes necrotic, and we see the unusual occurrence of a degenerated tissue remaining in an encased viable medium. The bacteria in this "incubator" cannot be attacked by the defense cells, for this mechanism can work only in the area where free exchange of tissue fluids is possible. The fluids enter the canal area but become static. In this way, they help to serve as a nutrient medium as well as another source of tissue irritation. The protein elements break down into the irritants which are in contact with the viable periapical tissue. This concentrated source of irritation, in conjunction with the bacterial toxins, may serve as the focus. The tissue is irritated, and, as the study by Fish[3] showed, the various zones of tissue reaction depend upon the concentration of the irritant. As the surrounding tissue becomes more receptive, the bacteria invade the new area. However, this status changes when the reaction of the tissue stabilizes. At this stage, the tissue passes from the acute state with an acid medium to a chronic alkaline medium. The bacteria are destroyed, and the reparative process is initiated.

An equilibrium is reached when the irritating factors from the canal are balanced by the defense mechanism of the periapical tissue. This quiescent state exists until either the irritant or the defensive tissue prevails. Unless completely removed, the source of irritation is always present; it acts as a force eliciting a reaction from the surrounding periapical tissue. It would therefore appear that repair can never take place while this focus remains.

This study seems to indicate that tissues which replace bone at the tooth apex following pulp degeneration can be considered one of the body defense mechanisms, that most of the roentgenolucent periapical areas are sterile, and that the occasional presence of bacteria is not of any practical significance.

SUMMARY

Is there any evidence of bacteria in periapical areas prior to the completion of root canal therapy? This question was investigated by making cultures of samples obtained from the apical areas. A modified Hedman technique was used. The canals were cleaned both mechanically and chemically prior to insertion of the cannula and stylet into the apex.

Although nineteen of the postreaming cultures were positive, only one of these gave a positive culture for the periapical area. The change in technical methods is assumed to have nearly eliminated the pushing of contaminants through to the apex. The sixty of the sixty-three cases which were periapically negative substantiated this contention. One case was positive throughout and might be considered a contaminant. In the second case, the prereaming and periapical cultures were positive, whereas the postreaming culture was negative. The third case was negative both pre- and postreaming and positive periapically.

These results lead one to the conclusion that most of the periapical areas are sterile and that the few cases that contain bacteria are of little statistical significance.

My appreciation and acknowledgement to Arthur H. Pearson, D.M.D., Professor of Endodontics, Chairman of the Department, Tufts University School of Dental Medicine, for his assistance in the organization and development of this study.

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Editor's Note: The reader may wonder why a paper which is predominantly endodontic in scope has been accepted for publication in the "Oral Roentgenology" section. The results of such investigations broaden the background of the oral roentgenologist and may motivate him to undertake similar but modified studies that have a yet stronger bearing on roentgenographic interpretation.

ROENTGENO-QUESTIONS

Is it advisable to have dental x-ray examinations while one is pregnant?

X-radiation can have no effect upon a fetus if the fetus is not exposed. Exposure of the fetus can be avoided in two ways: (1) by postponing the dental x-ray examination of the mother until after the birth of the baby or (2) by using a shield over the mother's abdomen. The use of a shield certainly can do no harm, even though it may be superfluous because of the small doses involved when proper techniques are used. The fetal exposure that results from a dental x-ray examination of fourteen exposures has been reported to be about 0.8 milliroentgen. The use of modern high-speed films and newer techniques could reduce this dose to less than 0.1 milliroentgen. From naturally occurring background radiation, the exposure of the mother and the fetus is about 0.3 milliroentgen per day. The significance of an additional 0.1 or even 0.8 milliroentgen of exposure in nine months has never been demonstrated clinically.

Does secondary radiation remain in the dental operating room after the primary x-ray beam is turned off?

All x-radiation in the room ceases the instant the primary beam is turned off.

The American Academy of Oral Roentgenology solicits from the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY any questions which deal with technical or interpretive aspects of oral roentgenology. These questions should be sent directly to the editor of this section, Dr. Arthur H. Wuehrmann, School of Dentistry, University of Alabama Medical Center, Birmingham, Alabama; the editor will distribute them for reply to a suitable group within the Academy. Questions, with the answers, will be published at as early a date as possible in "Roentgeno-Questions." Questions, especially those of an interpretive nature, should ordinarily be accompanied by good-quality roentgenograms and a pertinent history. The names of the questioner and the respondent will not be published.

ROENTGENO-ODDITIES

Roentgenopaque Mass in Incisive Foramen

IN RECENTLY reviewing an article by Dr. Karl Omnell in the July, 1959, issue of ORAL SURGERY, ORAL MEDICINE & ORAL PATHOLOGY, I was reminded of a case which appeared somewhat similar to that mentioned in Dr. Omnell's

Each month this section will bring to the readers of ORAL SURGERY, ORAL MEDICINE AND ORAL PATHOLOGY one or more roentgenograms which demonstrate unusual, unexpected, rare, or bizarre roentgenographic changes. These roentgenograms will be accompanied by an explanation or by words of inquiry regarding the particular change. All films used in this section will be accepted with the understanding that 2 by 2 or 3¼ by 4 inch slides or glossy photographs of the films will be made available to interested readers through the editor of the American Academy of Oral Roentgenology at the individual's expense. The cost will involve only the photographer's charges. Each slide or print will give credit to the donor. Interesting roentgenograms are solicited from all sources. Please be certain to identify your films properly so that they can be returned. All material for publication should be submitted to Dr. Arthur H. Wuehrmann, University of Alabama School of Dentistry, Medical Center, Birmingham, Alabama.

report. In the present case, which is illustrated on this page, the oral surgeon who removed the roentgenopaque mass described it as being "amalgam." The patient was unable to remember any instance of a dental office accident in which a mass of metallic material could have been forced into the palatal tissues. Roentgenographically, the opacity of this mass in the canal does not simulate amalgam which ordinarily would appear lighter. The roentgenographic evidence strongly suggests that this patient had suffered a blow which resulted in a fracture of the mesial incisal corner of the right central tooth. The vitality of the tooth at the time that this roentgenogram was made is not known. The case is simply submitted as a "Roentgeno-Oddity" suggesting that possibly the roentgenopaque mass in the incisive foramen is actually zinc carbonate and perhaps was formed in the same manner as that described in Dr. Omnell's article.

This case gives rise to interesting speculation. I would be interested in reader comment.

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ORAL PATHOLOGY

American Academy of Oral Pathology

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THE HISTOPATHOLOGY AND HISTOCHEMISTRY OF DERMATOLOGIC LESIONS IN THE MOUTH

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WHILE the histopathology of most of the more common dermatoses has been understood for many years, there is still considerable confusion regarding the microscopic appearance of the oral mucosal lesions of these dermatologic diseases. In the lesions of such diseases as lichen planus the histopathologic picture is often far from diagnostic or even suggestive, although for some years it has been considered a completely characteristic entity on which a diagnosis could be based even in the absence of suggestive clinical manifestations. In the lesions of such diseases as pemphigus, recent work has indicated that both biopsy and smear may be of diagnostic value. In other diseases, histochemical methods may be of considerable assistance in the evaluation of the biopsy specimen from a clinically confusing oral lesion.

We shall present our viewpoint on the histopathology of the oral lesions of lichen planus, chronic discoid lupus erythematosus, pemphigus vulgaris, and mucous membrane pemphigoid. We have been fortunate in having available a large amount of biopsy material with reference to these four dermatologic entities.

Concerning other common dermatoses, we have either not seen oral lesions or have studied too few of them under the microscope to be able to offer a reasonable viewpoint as to their histopathology.

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During recent years, in our biopsy studies of oral lesions it was felt that the use of various stains other than hematoxylin and eosin might provide further information and be of value in the differentiation of some of these lesions which may closely resemble one another when prepared with routine histologic stains. Among the various stains used, periodic acid-Schiff (P.A.S.) was of considerable value, both in localizing mucopolysaccharide substance in the tissues and in clarifying some of the structural abnormalities in mucosal lesions. The work of McManus[20, 21] and Lillie[18] pointed to the histochemical uses of periodic acid in localizing acid mucopolysaccharides. Altschuler and Angevine[2, 3] studied the distribution of mucopolysaccharides in degenerative diseases of connective tissue, and various dermatologic diseases were investigated histochemically for polysaccharides by Steiner,[24] Stoughton and Wells,[25] and others.[4, 6]

We now routinely stain all mucosal biopsy specimens representing oral dermatologic lesions with both hematoxylin and eosin and periodic acid-Schiff stains as well as a keratin stain developed in our laboratory by P. Ayoub (Mallory's connective tissue stain using 5 per cent acid fuchsin instead of 0.5 per cent). Keratin is brilliant red, prekeratin is orange, and epithelium is gray.

HISTORICAL REVIEW

A brief review of the literature on the histopathology of the common dermatoses with oral involvement is of interest.

Lichen Planus.—The histopathology of lichen planus was described with considerable clarity by Unna[28, 29] in 1894. In 1906 Dubreuilh[10] studied biopsy specimens from lesions of tongue and buccal mucosa and pointed out that the oral lesions were comparable in microscopic appearance to those of skin. He suggested the use of oral biopsy as a diagnostic aid because of the simplicity of the procedure. Trautmann,[26] in 1911, reviewed the subject of oral lichen planus from both a clinical and a histologic viewpoint. Substantial numbers of cases of oral lichen planus were reviewed by Cooke[9] in 1954 and by us[23] in 1961. We have pointed out that the oral lesions of lichen planus do not always present the classic microscopic features of the disease and that a diagnosis must be made on the basis of a correlation of clinical and microscopic evidence.

Chronic Discoid Lupus Erythematosus.—The histology of chronic discoid lupus erythematosus was reviewed by Kyrle[15] in 1909, Goeckeman and Montgomery[13] in 1932, and others.[26] Kyrle noted the transformation of collagenous tissue into a resorcinophilic mass but attached no great significance to it. The histology of the oral lesions was reviewed by Trautmann[26] in 1911 and by others.

Pemphigus.—The histologic features of pemphigus were discussed many years ago, but it was only recently, with the work of Civatte,[7, 8] Tzanck,[27] and Lever,[16] that the characteristic feature of acantholysis in pemphigus became well understood. Although for many years mucous membrane pemphigoid was considered a variant of pemphigus vulgaris and was referred to as pemphigus conjunctivae or benign mucous membrane pemphigus,[17] recent work by Lever[16] and by ourselves[22] has established this condition as a separate entity with histologic features quite distinct from those of pemphigus vulgaris and its group of variants.

MICROSCOPIC STUDIES

Lichen Planus.—The classic microscopic picture of oral lesions of lichen planus are parakeratosis, occasionally slight acanthosis and some downward extension of rete pegs, hydropic degeneration of the stratum germinativum, and an inflammatory infiltration of the connective tissue by a broad band of chronic inflammatory cells directly beneath the stratum germinativum of the epithelium. The chronic inflammatory infiltrate consists of lymphocytes with scattered histiocytes and occasional plasma cells. The deeper layers of the corium are relatively free of inflammatory infiltrate.

The increase in the stratum corneum may consist of a hyperkeratosis in many cases, rather than a parakeratosis (Fig. 1, *A*), and there may be a varying amount of acanthosis. There may be an area of hyperkeratosis adjacent to an area of parakeratosis. If the stratum corneum is sufficiently increased in width, the stratum granulosum will appear accentuated. The rete pegs may present some elongation, but the typical "saw-tooth" configuration seen in skin lesions is usually absent from mucosal lesions. In many instances the oral lesions present a generalized flattening of the rete pegs (Fig. 1, *A*).

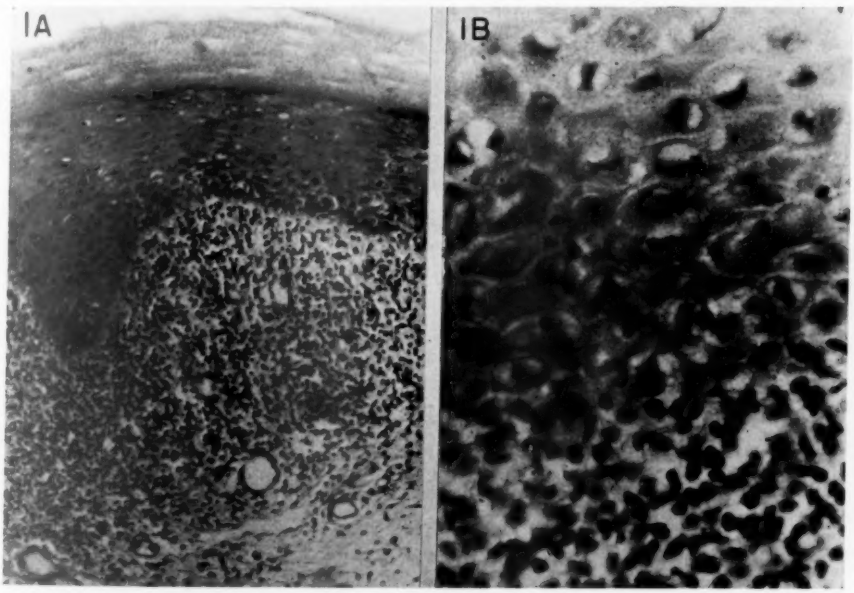

Fig. 1.—*A*, Medium-power photomicrograph of biopsy specimen showing lichen planus of the buccal mucosa. Note the extensive hyperkeratosis and the areas of hydropic degeneration, particularly in the stratum germinativum. The connective tissue is infiltrated by a zone of lymphocytes. However, the infiltrate is not situated directly adjacent to the epithelium, and the epithelium-connective tissue boundary is fairly well demarcated. There is a relative absence of rete pegs, rather than the classic saw-tooth hyperplasia. (Hematoxylin and eosin stain.)

B, High-power view of section shown in *A*. This field is at the epithelium-connective tissue boundary. Note the extensive hydropic degeneration of the epithelial cells, with cellular destruction and disappearance of the stratum germinativum. Note also the dense infiltration of lymphocytes and scattered plasma cells. (Hematoxylin and eosin stain.)

Dyskeratosis is not a feature of lichen planus. The hydropic degeneration of the stratum germinativum may involve a relatively small area, or it may affect the entire basal layer of the epithelium. The severity of the degenerative change may vary from cellular edema to complete destruction of the basal cells with consequent loss of architecture of the so-called basement membrane. These degenerative changes may involve cells of the inferior stratum spinosum as well as the cells of the stratum germinativum (Fig. 1, *B*). It must be understood that in many cases the hydropic degeneration is minimal, the basement membrane appears intact, and the architecture of the basal layer is normal (Fig. 2, *B*). In these cases there may be a slight amount of hyaline-like degeneration of the connective tissue adjacent to the basement membrane area.

The cellular infiltration is primarily lymphocytic in nature and is usually well localized as a broad band of cells lying adjacent to the epithelium and avoiding the deeper layers of the connective tissue. The infiltrate may be directly at the epithelium-connective tissue junction (Fig. 2, *A*), or there may be a narrow clear zone beneath the basement membrane (Fig. 2, *B*). In the former case the infiltrate often extends into the zone of epithelial degeneration, and the boundary between epithelium and connective tissue is indistinct. In the latter case the infiltrate is somewhat removed from the stratum germinativum, so that the boundary is well demarcated and obvious. It appears that this situation exists if the epithelial degeneration is minimal, whereas the presence of extensive hydropic degeneration permits the inflammatory infiltrate to extend up to and past the basement membrane area. Whether the basement membrane itself is a structure that undergoes lysis is doubtful, since it probably represents nothing more than a relative condensation or increased density of connective tissue ground substance. So-called degeneration of basement membrane is seen in many different inflammatory dermatoses.

With periodic acid-Schiff staining for mucopolysaccharides, there appears to be a well-outlined basement membrane area which is sharply defined but relatively thin. The inflammatory zone beneath the epithelium is well stained, as are the blood vessel walls and underlying connective tissue (Fig. 2, *C*). There is often an intense reaction for mucopolysaccharides in the area of the stratum germinativum. Fibrillar extensions are noted running from the basement membrane area into the underlying inflammatory zone (Fig. 2, *D*). The epithelial cells are relatively free of mucopolysaccharides, but the zone of parakeratosis often stains deeply.

Fig. 2.—*A*, Low-power study of lichen planus of buccal mucosa. Note the parakeratosis and acanthosis, as well as the very close approximation of the lymphocytic infiltration to the epithelium. The epithelium-connective tissue boundary presents an indistinct picture. (Hematoxylin and eosin stain.)

B, Medium-power photomicrograph from a case of lichen planus involving the buccal mucosa. Note the parakeratosis and slight hyperplasia. The lymphocytic infiltration appears as a broad band beneath the epithelium, but the epithelium-connective tissue boundary is distinct and well outlined. There is very little evidence of hydropic degeneration in the stratum germinativum, although some edema is noted in the stratum spinosum. (Hematoxylin and eosin stain.)

C, Medium-power photomicrograph of case of lichen planus. This is a serial section of the previous specimen, stained for mucopolysaccharides. Note the well-outlined basement membrane area. The inflammatory zone beneath the epithelium is well outlined, as are the blood vessel walls and underlying connective tissue. Note also the intense reaction in the area of the stratum germinativum. (Periodic acid-Schiff stain.)

D, High-power view of section shown in *C*. Note the sharp line representing the basement membrane. Extensions from this line into the underlying inflammatory reticulum can be seen. The lymphocytic cells are moderately stained, while the epithelial cells are relatively free of mucopolysaccharides. (Periodic acid-Schiff stain.)

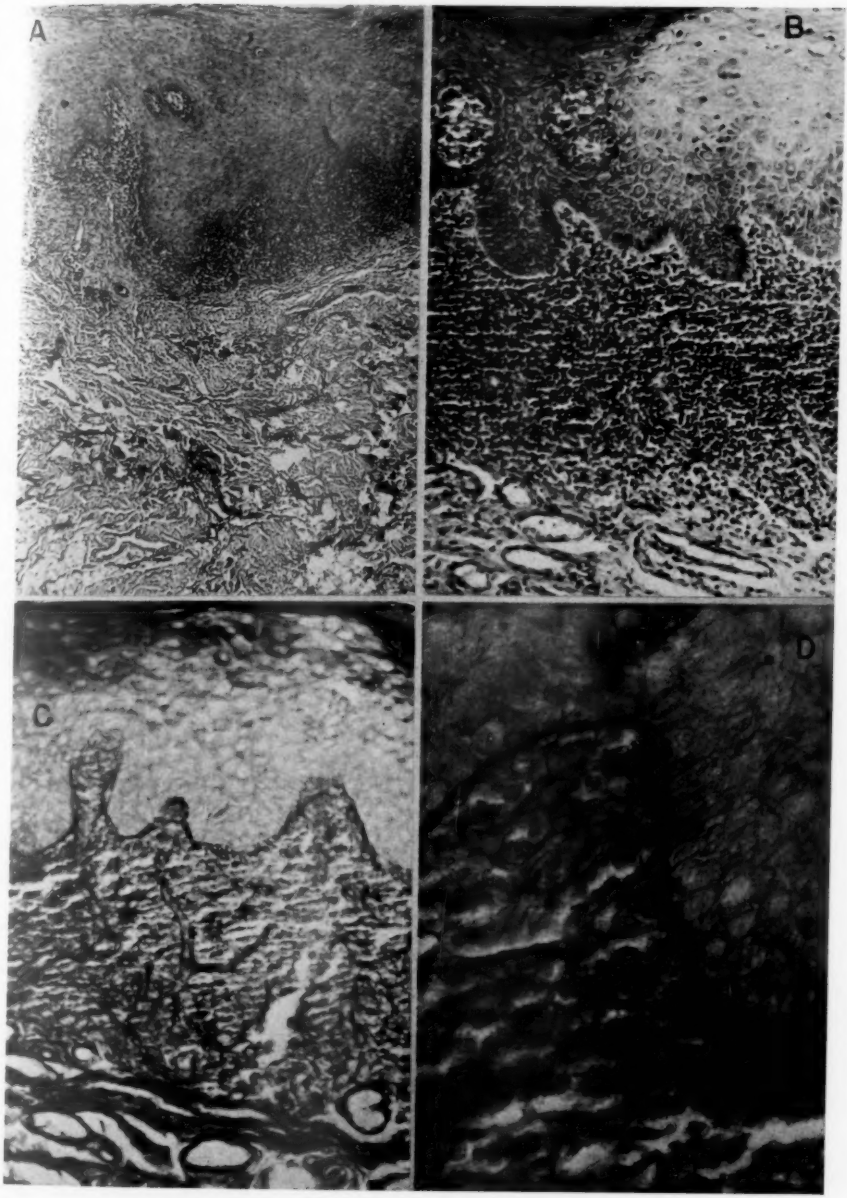

Fig. 2.—*(For legend, see opposite page.)*

In summary, for purposes of microscopic diagnosis there is a triad of (1) parakeratosis or hyperkeratosis, (2) hydropic degeneration of the basal layer of the epithelium, and (3) chronic inflammatory infiltrate in the configuration of a wide band adjacent to and beneath the epithelium.

In relation to the histology of oral lichen planus, several points must be emphasized. Lichen planus cannot be diagnosed on the basis of histopathologic findings alone. There must be a correlation of both clinical and microscopic evidence. The oral lesions of lichen planus often present a microscopic picture of nonspecific chronic inflammation.[23] This picture may change during the course of the disease to include a more classic group of microscopic features, or in certain cases it may remain nonspecific throughout the course of the lesion.

Histochemical evaluation has not helped us in the diagnosis of lesions with a nonspecific microscopic appearance.

Chronic Discoid Lupus Erythematosus.—The classic picture of the oral lesions of chronic discoid lupus erythematosus consists of a moderate parakeratosis or hyperkeratosis, hydropic degeneration of the stratum germinativum, degeneration of the connective tissue collagen, and an infiltration of chronic inflammatory cells. The cellular infiltrate is primarily lymphocytic and tends to be arranged in a perivascular pattern (Fig. 3, *A*). In skin lesions the lymphocytic infiltration is seen in localized clusters which may or may not surround small blood vessels. In oral lesions the arrangement is invariably perivascular, but there may be an extremely dense infiltrate and the perivascular pattern is here obscured. This is particularly true in the case of gingival lesions where secondary gingival and periodontal involvement may accentuate the inflammatory picture.

There still appears to be some doubt concerning the question of collagen degeneration in the skin lesions of chronic discoid lupus erythematosus. We feel that collagen degeneration is a constant feature in the histopathologic picture of the oral lesions of chronic lupus erythematosus. The degeneration is seen most clearly in the corium directly adjacent to the epithelium. The connective tissue substance appears as a homogeneous, hyaline-like substance which

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Fig. 3—*A*, Medium-power section of biopsy specimen from a case of chronic discoid lupus erythematosus involving the buccal mucosa. Note the hyperkeratosis and the hydropic degeneration of the stratum germinativum. Beneath the epithelium there is a relatively clear zone of hyaline-like material. Note the chronic inflammatory infiltration into the underlying connective tissue. The infiltrate tends to be perivascular (*BV*) or to spread homogeneously throughout the connective tissue. (Hematoxylin and eosin stain.)

B, High-power view of section shown in *A*. Note the well-defined stratum granulosum beneath the stratum corneum. The stratum germinativum presents hydropic degeneration, with many nuclei being absent. Note the coagulated hyaline-like substance beneath the epithelium, representing degeneration of the connective tissue collagen. (Hematoxylin and eosin stain.)

C, Medium-power view of biopsy specimen from a case of lupus erythematosus involving the buccal mucosa, stained for mucopolysaccharides. Note the relative absence of stain in the epithelium and the extremely intense reaction in the corium immediately adjacent to the epithelium. Note also the reaction of some collagen bundles and some blood vessels. The intense stain beneath the basement membrane represents the hyaline-like areas in the hematoxylin and eosin preparations. (Periodic acid-Schiff stain.)

D, High-power survey of a case of oral lichen planus stained for mucopolysaccharides. Note the outline of the basement membrane and the absence of the intense connective tissue reaction as seen in lupus erythematosus. (Periodic acid-Schiff stain.)

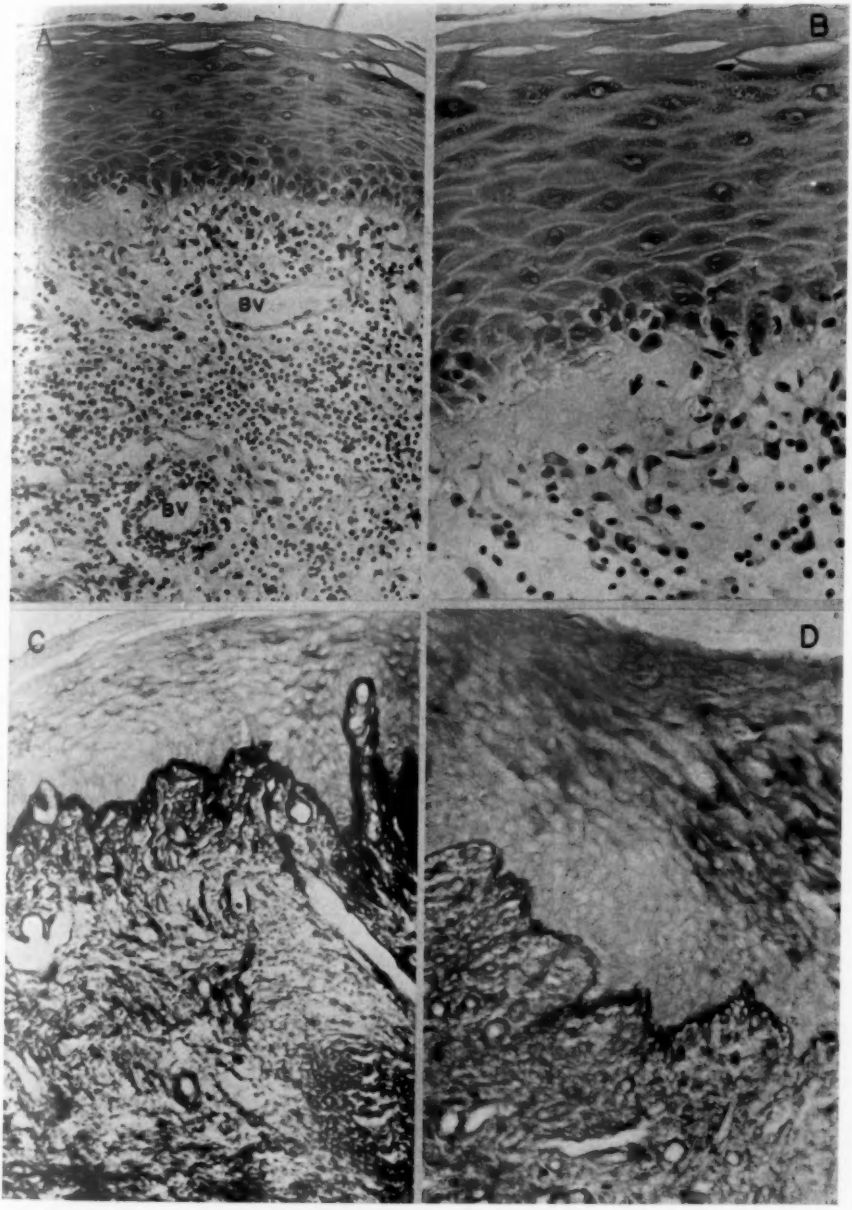

Fig. 3.—*(For legend, see opposite page.)*

stains light pink with hematoxylin and eosin (Fig. 3, *B*). This type of substance is also seen around small blood vessels. The remainder of the connective tissue appears relatively devoid of fibrillar material and of the pale-staining ground substance.

The stratum germinativum of the epithelium presents areas of degeneration ranging from hydropic degeneration to complete cellular disintegration. In this respect, lesions of chronic lupus erythematosus often bear a histologic resemblance to those of lichen planus, since both are also hyperkeratotic lesions. The hyperkeratosis in chronic lupus erythematosus is not usually so marked as in lichen planus, and consequently the oral lesions are not so white clinically as a rule.

P.A.S. staining for mucopolysaccharides appears to be of great value in diagnosing the oral lesions of chronic lupus erythematosus. The characteristic finding is an extremely intense reaction beneath the stratum germinativum of the epithelium and around small blood vessels (Figs. 3, *A* and 4, *B* and *C*). These areas of intense reaction to staining for mucopolysaccharides represent the areas of collagen degeneration—the hyaline-like substance noted in the sections stained with hematoxylin and eosin (Fig. 4, *A* and *B*). The stratum granulosum and occasionally the stratum corneum present some reaction when stained for mucopolysaccharides, but the striking change is in the connective tissue adjacent to the epithelium. The stain is considerably more intense than that seen in cases of lichen planus in the same area. The zone of reaction is much wider, and the reaction is also noted around vascular channels.

In summary, the microscopic features of the oral lesions of chronic discoid lupus erythematosus are (1) hyperkeratosis or parakeratosis, (2) hydropic degeneration of the stratum germinativum, (3) a hyaline-like degeneration of connective tissue collagen, and (4) a lymphocytic infiltration arranged in clumps and usually in a perivascular pattern. P.A.S. staining for mucopolysaccharides evokes an intense reaction in the areas of collagen degeneration. This latter finding appears to be reasonably characteristic of chronic lupus erythematosus and has been noted in all cases studied. An examination of a large number of cases may establish this conclusively.

Lichen Planus Bullosa.—Lichen planus bullosa represents the characteristic features of lichen planus with accentuated hydropic degeneration of the epithelial cells of the stratum germinativum and lower stratum spinosum. The severity of the reaction results in the accumulation of a considerable amount of fluid at the epithelium-connective tissue junction, with the lifting-off of the remaining overlying epithelium and the formation of a subepithelial vesicle (Fig. 5, *A* and *B*). The connective tissue is densely infiltrated with chronic inflammatory cells. As the vesicle ruptures, there is an ulcerated area complicated by oral infection, so that large numbers of polymorphonuclear leukocytes are in evidence at the surface of the exposed connective tissue. Hemorrhage is also a feature. This lesion could be considered erosive or ulcerative lichen planus.

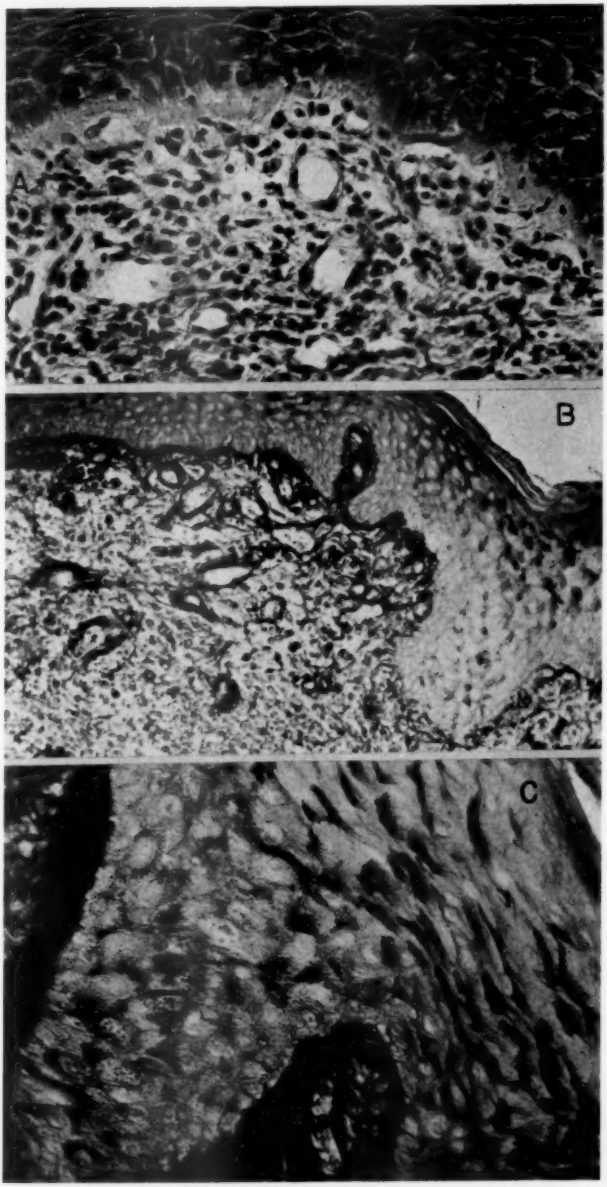

Fig. 4.—*A*, High-power photomicrograph of section from an oral lesion of lupus erythematosus. Note the hyaline-like zone beneath the epithelium and the generalized infiltration of lymphocytes. (Hematoxylin and eosin stain.)

B, Medium-power view of specimen from lesion shown in *A*, stained for mucopolysaccharides. Note the intense reaction in this hyaline-like zone of degeneration. Note also the intense reaction around some blood vessels. (Periodic acid-Schiff stain.)

C, High-power study of same case. Note the intense reaction for mucopolysaccharides in the connective tissue. Note also the stain within some of the epithelial cells. (Periodic acid-Schiff stain.)

With P.A.S. staining, the deeper layers of the corium stain intensely (Fig. 6, *A*) and the basement membrane zone stains strongly for mucopolysaccharides. The stratum corneum stains deeply.

In lichen planus bullosa the lesions at the periphery of the vesicle may present a classic microscopic picture of lichen planus.

Pemphigus Vulgaris.—For many years the histopathologic picture of pemphigus vulgaris was considered to be nonspecific. However, the works of Civatte,[8] Tzanck,[27] and Lever[16] have pointed to a definite and characteristic microscopic picture. The histologic appearance of the oral lesions is entirely similar to that of the skin lesions and often more clear-cut for diagnostic purposes.

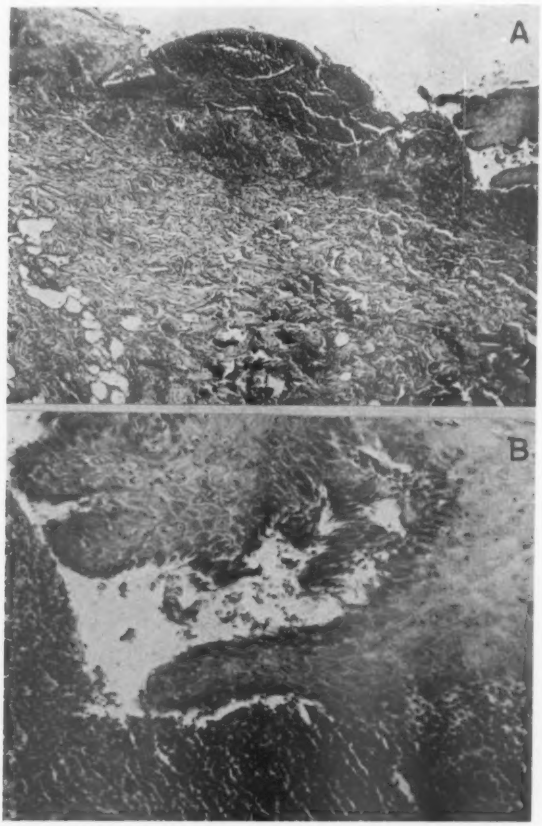

Fig. 5.—*A*, Low-power photomicrograph of section from an oral lesion in a case of bullous lichen planus. Note the break in the epithelium and the central area of ulceration with hemorrhage. The connective tissue is densely infiltrated with inflammatory cells. (Hematoxylin and eosin stain.)

B, High-power photomicrograph of section at margin of lesion shown in *A*. Note the separation of the epithelium from the underlying connective tissue with the formation of a subepithelial vesicle. (Hematoxylin and eosin stain.)

The basic alteration is acantholysis. There is a split within the epithelium, usually in the stratum spinosum just above the stratum granulosum. However, the split may occur higher up in the stratum spinosum. There is then a separation at the intraepithelial split, with formation of an intraepithelial vesicle (Fig. 7, *A*). As fluid collects in this space, the vesicle increases in size. The cells of the stratum spinosum appear to undergo degeneration. They lose their adhesiveness and fall apart, possibly because of shriveling of the protoplasmic tonofibrils and a dissolution of the intercellular cementing substance. These cells with large, deeply staining nuclei (Fig. 7, *B*) are often seen lying free in the vesicle. They can be smeared from a ruptured vesicle and may be utilized in the cytodiagnosis of pemphigus.[27] In addition to these acantholytic epithelial

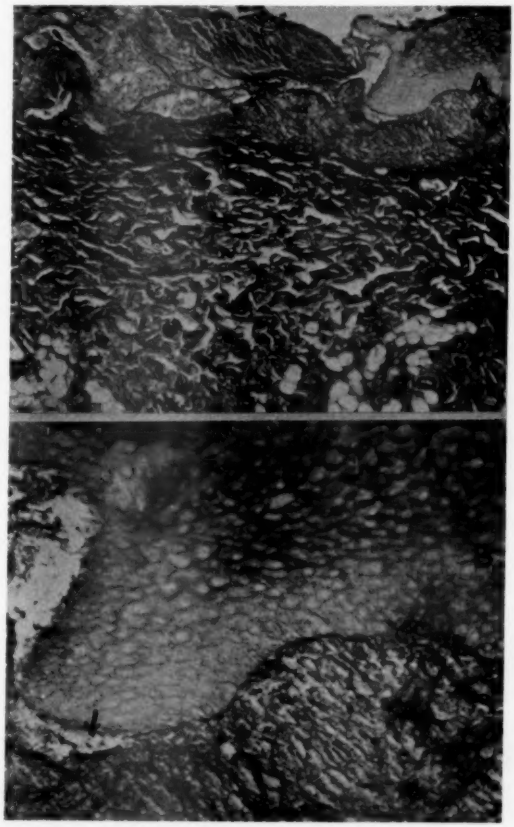

Fig. 6—*A*, Low-power photomicrograph of specimen from same case as shown in Fig. 5, stained for mucopolysaccharides. Note the rather intense reaction in the deeper layers of the corium. (Periodic acid-Schiff stain.)

B, High-power study of specimen taken at margin of lesion. Note the gradual separation of epithelium from underlying connective tissue. Note also the staining of the basement membrane zone and the suggestion that this also separates from the epithelium. The cells of the stratum spinosum take a granular stain and the stratum corneum stains intensely. (Periodic acid-Schiff stain.)

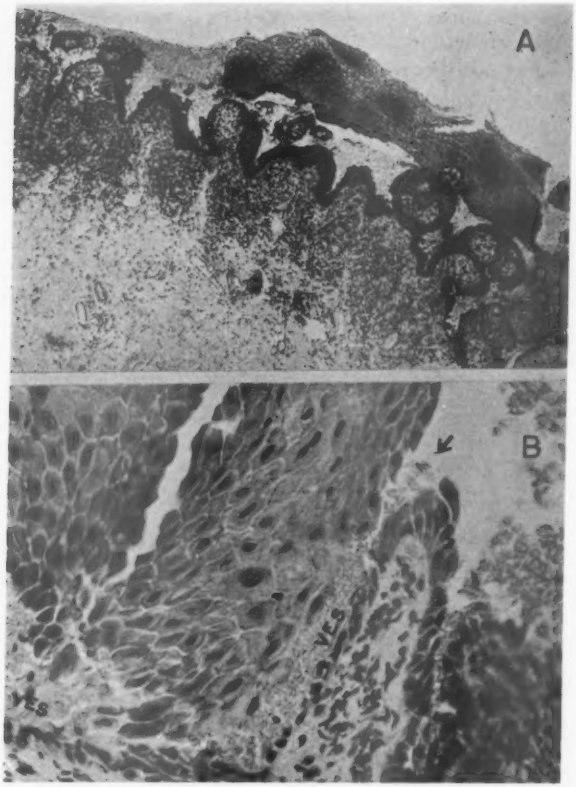

Fig. 7.—*A*, Low-power study of section from an oral mucosal lesion of pemphigus vulgaris. Note the separation of the lower layers of the epithelium from the bulk of the stratum spinosum, with the formation of an intraepithelial vesicle. (Hematoxylin and eosin stain.)

B, High-power photomicrograph of an oral vesicle of pemphigus vulgaris. Note the splitting-off of the stratum germinativum from the stratum spinosum (arrow). Note also the formation of an intraepithelial vesicle (*VES*) and the lack of cohesion of the epithelial cells which appear to be separating and entering the vesicle (acantholysis). (Hematoxylin and eosin stain.)

cells, the vesicle contains fluid, erythrocytes, and scattered inflammatory cells. The underlying connective tissue is densely infiltrated with lymphocytes, plasma cells, histiocytes, and polymorphonuclear leukocytes. Eosinophils are seen occasionally and vary in quantity.

When the vesicle reaches microscopic size and ruptures, the lesion is complicated by oral infection and the remaining epithelial cells of the basal layer are destroyed, leaving an ulcer. Biopsy specimens must always be taken at the margin or periphery of a vesicle or ulcer of pemphigus vulgaris.

In summary, the acantholysis, splitting, and peeling-off of the upper layers of the epithelium (Fig. 7, *B*) are characteristic of pemphigus vulgaris. The ease with which oral biopsy procedures are carried out may point to this technique as an important diagnostic aid in a questionable case of pemphigus. Cytologic diagnosis may be helpful in cases of pemphigus vulgaris. We have not

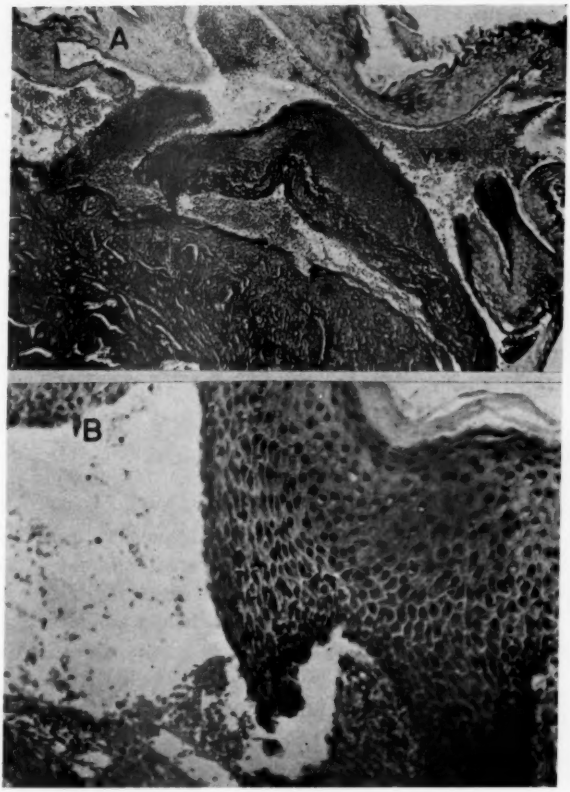

Fig. 8.—*A*, Low-power study of an oral vesicle of mucous membrane pemphigoid stained for mucopolysaccharides. Note the lifting-off of the entire epithelium, with the formation of a large subepithelial vesicle. Note also the stain in the stratum corneum of the epithelium and the outline of the basement membrane. (Periodic acid-Schiff stain.)

B, High-power study of section from the margin of an oral vesicle of mucous membrane pemphigus. Note the clear-cut separation of the stratified squamous epithelium from the underlying connective tissue. There is no evidence of acantholysis as in pemphigus vulgaris. (Hematoxylin and eosin stain.)

had sufficient experience with this procedure. We have not had occasion to study a single case of oral pemphigus vegetans or oral pemphigus foliaceus, and we tend to doubt their existence. These variants refer to skin lesions. Oral lesions, when present in these cases, are of the typical pemphigus vulgaris variety.

P.A.S. stains have not added to our knowledge of the histology of pemphigus vulgaris.

Mucous Membrane Pemphigoid.—This condition was formerly considered to be a variant of pemphigus, but studies by Lever[16] and by us[22] have established it as a clinical entity separate from pemphigus and characterized by a different histologic picture. Thus, the term "pemphigoid" is now employed. The oral lesions of mucous membrane pemphigoid show no evidence of acantholysis, no intraepithelial vesicle formation, and no separation or falling-apart of cells of

the stratum spinosum. The vesicle is subepithelial, and the split is at the epithelium-connective tissue margin. The entire epithelium is lifted off from the underlying connective tissue (Fig. 8, *A*). The vesicle contains fluid, erythrocytes, and scattered inflammatory cells. The underlying connective tissue is infiltrated with lymphocytes, plasma cells, histiocytes, and polymorphonuclear leukocytes. P.A.S. staining does not contribute to our knowledge of this disease. The so-called basement membrane stains for mucopolysaccharides and appears to come along with the epithelium as it is lifted off from the connective tissue (Fig. 8, *A*). As the vesicle ruptures, we see an ulcer which becomes involved by oral infection.

In summary, the histopathology of mucous membrane pemphigoid is a nonspecific picture of subepithelial vesicle formation. It resembles the vesicle of erythema multiforme and other nonspecific lesions. Eosinophils are occasionally seen in mucous membrane pemphigoid lesions, but they are not so striking a feature as in erythema multiforme.

DISCUSSION

According to Allen,[1] the histologic picture of lichen planus is diagnostic on the basis of the following features: (1) hyperkeratosis, (2) prominent stratum granulosum, (3) acanthosis, (4) elongated, pointed ("saw-toothed") rete pegs, (5) liquefaction degeneration at the junction of the epidermis and dermis, (6) the absence of spongiosis, (7) a conspicuous, compact zone composed chiefly of lymphocytes and histiocytes, with a sprinkling of eosinophilic and neutrophilic leukocytes on occasion, closely apposed to the epidermis and sharply delimited to the upper cutis, and (8) melanin-laden chromatophores just beneath the inflammatory area.

Our impression concerning the oral lesions of lichen planus is that the microscopic features do not always represent the classic picture and that the histologic picture may be suggestive but is not sufficient to be diagnostic. Diagnosis of oral lichen planus must be based on a correlation of clinical and microscopic evidence. The oral lesions seldom present the features of saw-toothed rete pegs, and they do not present the melanin-laden chromatophores. Similarly, hydropic degeneration is not always a striking feature of the oral lesions. Thus, the histopathology of oral lichen planus is not so specific as previously believed.

The histologic features of chronic lupus erythematosus involving skin are described by Allen[1] as (1) more or less alternating acanthosis and atrophy, (2) focal parakeratosis and hyperkeratosis, (3) keratinous follicular plugging, (4) liquefaction degeneration at the epidermis-dermis junction, (5) superficial dermal telangiectasis, and (6) dense collections of lymphocytes, particularly in the middermis and without quantitative relationship to the degree of follicular plugging or comedo formation. These foci of infiltration, which have much diagnostic importance, need have no relation to the appendages, despite what is commonly recorded to the contrary. Moreover, degeneration of collagen or of the walls of vessels is not considered to be a part of the process of chronic lupus erythematosus.[1] Lever[17] feels that in most instances a histologic diagnosis is

possible on the basis of a combination of pathologic findings. He reports the five cardinal changes in the skin as follows: (1) hyperkeratosis with keratotic plugging, (2) atrophy of the rete malpighii, (3) liquefaction degeneration of the basal cells, (4) a patchy, perivascular, chiefly lymphocytic infiltrate with a tendency to arrangement about the dermal appendages, and (5) basophilic degeneration of the collagen and of elastic fibers. Lever stresses, however, that not all five changes are present in every case. The collagen degeneration is also noted by Gans and Steigleder.[11] A study of the oral lesions appears to point to certain differences, but the general pattern resembles that of skin lesions. The keratinous follicular plugging is, of course, a feature of skin lesions only. The foci of chronic inflammatory infiltration appear to us to be perivascular in nature. Furthermore, there is little doubt as to the existence of collagen degeneration. This is demonstrated particularly well by periodic acid-Schiff staining, and this striking reaction may well be diagnostic for the oral lesions of chronic lupus erythematosus.

Our studies on pemphigus vulgaris corroborate those of Civatte[8] and Lever.[16] The oral lesions all present the characteristic acantholysis. We stress that biopsy specimens must be taken carefully at the periphery of lesions relatively uncomplicated by oral infection and trauma. Since lesions of pemphigus vulgaris often appear first on the oral mucosa, biopsy study may prove an extremely important diagnostic aid. Cytodiagnosis of acantholytic cells may be possible, but our experience has been limited in this diagnostic procedure with reference to pemphigus.

Mucous membrane pemphigoid presents a nonspecific picture of subepithelial vesicle formation and can thus be distinguished from pemphigus vulgaris. Histologically, however, there may be some similarity between mucous membrane pemphigoid and bullous lichen planus. Clinical correlation thus becomes extremely important.

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ORAL PATHOLOGY

General Section

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INFANTILE CORTICAL HYPEROSTOSIS OR FIBROUS DYSPLASIA

A Diagnostic Problem

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In 1939 Caffey[1] reported the case of an infant with scattered swellings in several bones. The cortical thickening beneath the periosteum could not be readily explained on the basis of scurvy, rickets, syphilis, osteomyelitis, osteitis, or neoplasm. From 1939 to 1944 three similar cases were reported by Smyth at the University of California. Three findings were similar in both sets of cases: (1) onset during the first three months of life, (2) tender swellings deep in soft tissues, and (3) cortical thickening in the skeleton. Both groups of cases presented similar lesions of cortical hyperplasia under a proliferating periosteum. The bones affected in the order of frequency, were the mandible, the clavicle, the scapula, the ribs, and the extremities. There was no evidence of pitting edema or other signs of inflammation caused by infection. Lymphadenopathy was strangely absent in all cases.

CLINICAL COURSE

The edema of the affected part appeared and regressed a number of times during the process without any change in the x-ray appearance of the lesion. Some patients showed an elevation of temperature, whereas others developed pleurisy and anemia. All recovered after various periods of time. It was observed that x-ray evidence of the bone lesion may not become positive for four to six weeks after onset. Pleurisy usually was found when there was cortical thickening of the ribs.

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MICROSCOPIC FINDINGS

There were no signs of subperiosteal hemorrhage or cellular inflammation. The only demonstrable lesion was simple cortical hyperplasia. Cultures proved negative, and therefore antibiotic and vitamin therapy did not materially affect the clinical course of the disease.

Some symptoms were not present in all cases. However, fever, hyperirritability, pseudoparalysis, dysphagia, pleurisy, anemia, leukocytosis, monocytosis, increased sedimentation rate, and elevated alkaline phosphatase were common.

DIFFERENTIAL DIAGNOSIS

Facial swelling early in the disease suggests parotitis, osteomyelitis, or neoplasm. Scurvy exhibits symptoms of painful pseudoparalysis, swelling, and tenderness. Poliomyelitis may show symptoms of hyperirritability and pseudoparalysis. Bone pain and anemia are common in infantile leukemia. Rheumatoid arthritis and rheumatic fever are characterized by fever, pain, tenderness in the extremities, and elevated sedimentation rates.

CASE REPORT

On Feb. 14, 1955, an 8-year-old white boy was presented for diagnosis and treatment of a swelling in the body of the right mandible. The child, who did not sleep well and was restless and irritable, complained of continual pain in the right mandibular area.

History.—The boy's parents stated that the swellings had occurred monthly for the past six months. Discussion with the pediatrician elicited a history of inflammatory mandibular and long-bone lesions in infancy which Dr. John Caffey had diagnosed as infantile cortical hyperostosis. Dr. Caffey had also considered the possibility of infantile scurvy. There had been no recurrence of the disease prior to the present complaint.

Examination.—X-ray examination (Fig. 1) revealed an osteoporosis in the body and lower border of the mandible which suggested osteomyelitis. The oral temperature was 100° F., and the right mucobuccal fold of the mandible was noticeably expanded. This area was tender to palpation and showed some pitting on pressure. The teeth were in good repair, and all necessary teeth were present.

Course of Treatment.—In view of the clinical picture, the tentative diagnosis of osteomyelitis was considered. Antibiotic therapy over a period of several days did not relieve the pain. Incision for drainage and relief of the edema was now considered necessary, and the child was admitted to the hospital. A preoperative culture was obtained under sterile conditions. The area was then incised from the right molar region to the left premolar area, and the opening was packed with iodoform. The culture proved negative for any growth but, as might be suspected, the sedimentation rate and the alkaline phosphatase level were elevated.

In view of these findings, the diagnosis of pyogenic osteomyelitis was ruled out and the possibility of bone tumor was considered. Consultation resulted in the agreement that this was not a case of early bone tumor (sarcoma, etc.). A biopsy of the bone lesion seemed to be the next logical step, but this was deferred because the presence of teeth in the jaws would necessitate a deforming operation. (This problem, incidently, had arisen in many cases of patients afflicted with infantile cortical hyperostosis. In fact, biopsy or excision procedures had left many children crippled for life.) In any case, after a discussion with Dr. Caffey, it was decided that some time should be allowed to elapse for observation before any surgical intervention was considered. Several weeks later x-ray examination disclosed a progressive bone healing with regression of all symptoms (Fig. 2). However, the thickness of the body of the mandible did not regress.

The child remained symptom-free until Aug. 15, 1955, when the temperature rose to 101.8° F. and the right submaxillary triangle became tender and swollen. Another course of antibiotics was given, and the temperature subsided two days later. The remainder of

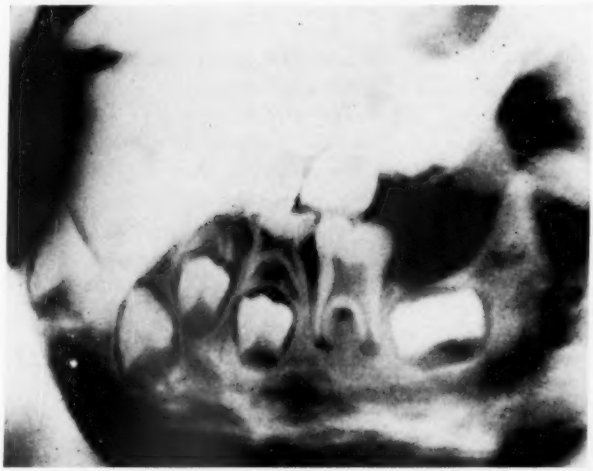

Fig. 1.—Feb. 14, 1955. Osteoporosis of body of mandible with periostitis.

Fig. 2.—March 17, 1955. Regression of bone lesions noted.

the examination yielded essentially the same findings as the first one. During the next three years there were no less than twelve additional episodes. It is interesting to note that each time that the radiolucency and periosteal thickening occurred these lesions regressed to normal roentgenographic appearance before the next attack. During this period the right body and the symphysis of the mandible remained enlarged (Fig. 3). In 1960 comparison of the intraoral roentgenograms (Fig. 4) with those of a normal child the same age (Fig. 5) showed a striking demineralization and ground-glass appearance consistent with a replacement of some

of the bone architecture with fibrous tissue. Throughout the clinical course of this case the only positive laboratory finding was an increased alkaline phosphatase level. This, of course, is consistent with any bone activity of this type.

CONCLUSIONS

In such cases as the one just described caution in treatment is imperative, for it is difficult to evaluate a process which obviously is inflammatory and does respond to Meticorten therapy. The clinical course of the disease suggests infantile cortical hyperostosis, but the later x-ray findings strongly suggest fibrous dysplasia. If the lesion is compared to some of the criteria for fibrous dysplasia, the great similarity between these diseases will immediately become apparent. The disease begins in childhood; calcium and phosphorus levels of the blood are not affected; the alkaline phosphatase level is usually normal, or

Fig. 3.—Roentgenogram taken on Jan. 23, 1958, when ground-glass appearance of mandible was first noted.

it may be elevated during the active phase of the disease. Most of the lesions of fibrous dysplasia are self-limiting and even have a tendency to regress after the patient reaches maturity.

The case just described presented the difficult diagnostic problem of a bone lesion which obviously was related to a systemic disorder. The original diagnosis of infantile cortical hyperostosis made when the patient was an infant may be misleading if one tends to overlook the possibility of a new or different disease process which developed when this same patient was 8 years of age. Roentgenograms taken several years later reveal the ground-glass appearance of the lesion of fibrous dysplasia, that is, one which shows replacement of some calcified elements with other tissues. The lack of biopsy material makes it difficult to come to any final conclusion in this case. However, the lesion now

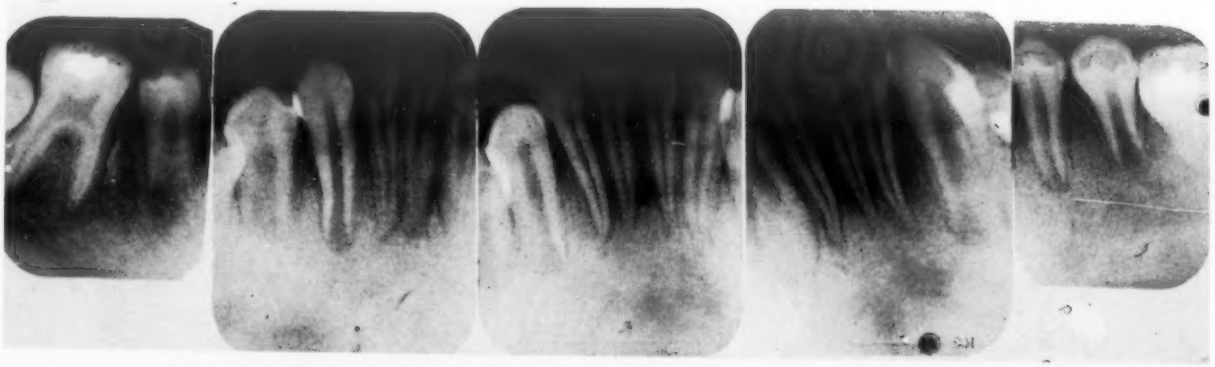

Fig. 4.—Intraoral roentgenograms taken on Feb. 10, 1960, showing ground-glass radiopacity of bone.

Fig. 5.—Intraoral roentgenograms of normal 10-year-old child. Compare with Fig. 4.

strongly suggests fibrous dysplasia rather than a rare case of infantile cortical hyperostosis in a 12-year-old child. The age factor alone should tend to rule out the earlier diagnosis.

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1927-31 MOTT AVE.

FIBROSARCOMATOUS EPULIS

Report of a Case

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THE following case is reported because of the rarity of the condition and because certain atypical features were found.

CASE REPORT

The patient, a 40-year-old schoolmaster, was referred by his general dental practitioner on July 22, 1959, for evaluation and treatment of swelling of about one year's duration lying buccally in the region of the lower left premolars. At no stage had there been any severe pain, but at times the patient had noticed some soreness and swelling of the cheek.

Fig. 1.—Clinical appearance.

He commented that he had noticed a "lump beneath the jaw" which arose soon after the intraoral swelling. The patient was an apparently fit man of average physique. On palpation, a stony-hard swelling could be felt in the left submandibular triangle. This did not appear to be attached to skin or to any of the deeper tissues.

Intraoral examination disclosed a mouth with some caries, chronic gingival inflammation, and calculus, requiring routine treatment. Signs were present of the sockets of the lower left lateral incisor and second molar, these teeth having been extracted a month previously. It was also noticed that the lower left second premolar was rotated. There was a tender, sessile swelling which measured about 1 inch in diameter and was pale in color except for a red zone surrounding a central sinus from which pus could be expressed on pressure. This swelling appeared to arise from the buccal mucoperiosteum of the alveolar process in the lower left premolar region (Fig. 1). The lining of the sinus appeared to consist of a yellowish white slough. The lower left canine and premolars gave vitality responses within normal limits.

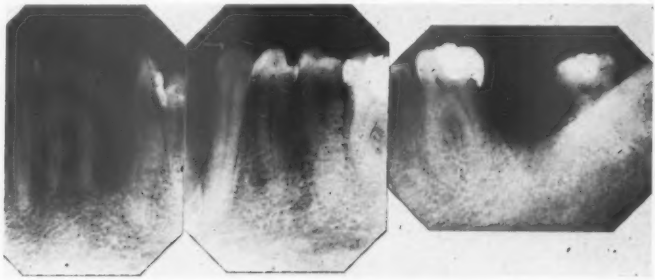

Fig. 2.—Roentgenograms of affected area.

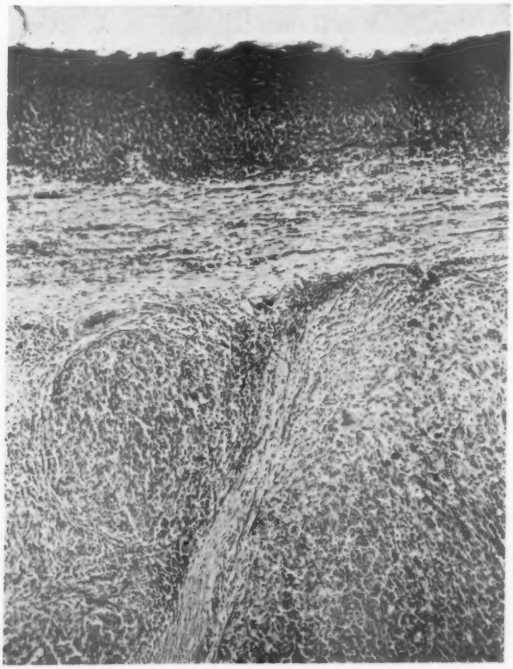

Fig. 3.—Section of tumor showing surface zone. (Hematoxylin and eosin stain. Green filter. Magnification, ×90; reduced ¼.)

Radiographic examination (Fig. 2) disclosed healing sockets in the region of the lower left lateral canine and second molar but no significant abnormality in the region of the lower left canine and premolars other than a slight horizontal loss of crestal bone.

A provisional diagnosis of unspecified neoplasia was made, but, in view of the hardness of the submandibular swelling, it was thought probable that the lesion was carcinomatous. We therefore decided to remove tissue for histologic examination at this visit.

The swelling, together with a surrounding zone of clinically normal mucosa, was removed under block anesthesia. This shelled away from the mandible without difficulty or excessive bleeding, leaving an apparently clean bone surface. The swelling was found to consist of a firm, white homogeneous mass with a central soft, almost caseous, area. The specimen was fixed in formal calcium, dehydrated with butyl alcohol, and embedded in paraffin. Sections were cut at 7 microns and stained by various techniques, including hematoxylin and eosin stain and hematoxylin and van Gieson's stain.

Histologically, the tumor was covered by parakeratotic oral epithelium beneath which was found a layer of white fibrous connective tissue in which most of the fibers were running parallel to the surface, forming a pseudocapsule. A few chronic inflammatory cells were found between the fibrous-tissue bundles and scattered diffusely beneath the epithelial surface (Fig. 3). The main bulk of the tumor was made up of large spindle cells arranged in bundles, showing many mitotic figures and interlaced with strands of white fibrous tissue (Figs. 4 and 5). Only a few blood vessels could be seen, and a zone of necrosis corresponding to the clinical "sinus" was found in the center of the lesion.

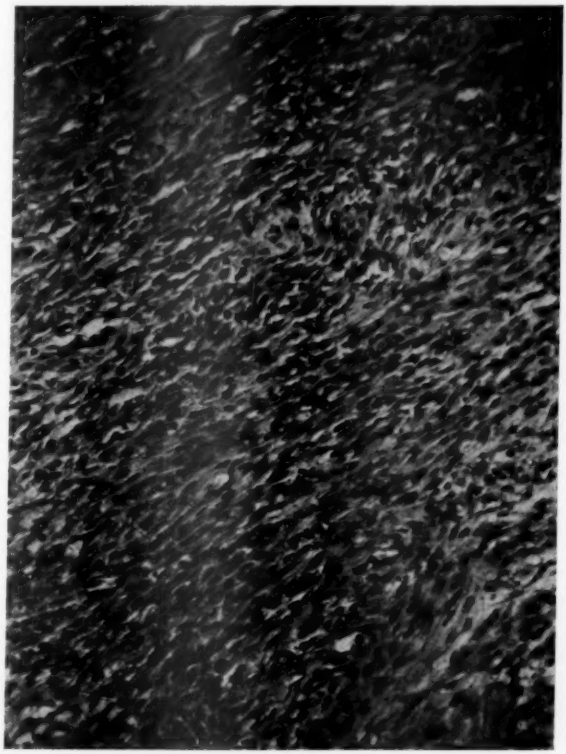

Fig. 4.—Section of main tumor mass showing cell arrangement. (Hematoxylin and eosin stain. Green filter. Magnification, ×250; reduced ¼.)

In view of the histologic findings, the tumor was diagnosed as a very anaplastic fibrosarcoma and arrangements were made for the patient to be hospitalized immediately for further investigation and surgery in the plastic and oral surgery unit.

Fig. 5.—Detail of section shown in Fig. 4 showing many mitotic figures. (Heidenhain's iron hematoxylin and eosin stain.)

Fig. 6.—Decalcified section of root of lower left second premolar. (Hematoxylin and eosin stain. Green filter. Magnification, ×180; reduced ¼.)

Additional examinations, including roentgenographic studies of the skeleton and chest, showed no sign of metastasis. There remained the possibility that the submandibular swelling might be the site of a secondary lesion.

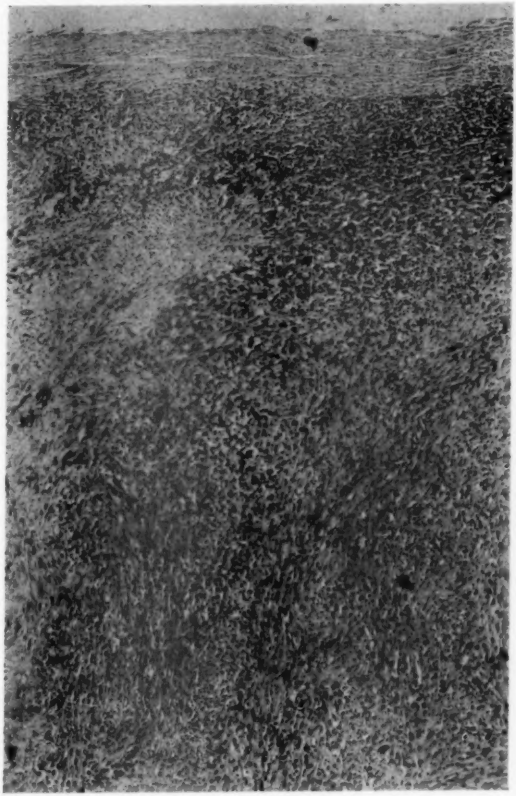

Fig. 7.—Section of submandibular lymph node showing encapsulation and replacement of normal lymphoid tissue. (Hematoxylin and eosin stain. Green filter. Magnification, ×80; reduced ¼.)

On July 29 the mandible with adjacent buccal soft tissue was resected from the left angle to the symphysis, and the left suprahyoid lymph nodes and the submandibular swelling were removed.

Examination of the excised specimens showed an ulcer at the site of operation, with an underlying depression in the bone adjacent to the root of the lower left second premolar. The root of the tooth showed an area of resorption which did not appear to be associated with multinucleated giant cells (Fig. 6). Tumor tissue appeared to fill the resorbed area. The tumor mass lining the ulcer was made up of fairly plump spindle-shaped cells showing much mitotic activity and arranged generally in parallel and interlacing bundles. The submandibular swelling was found to be a lymph node attached to the periosteum and lying between the mandible and the submandibular salivary gland. Histologically, this was found to be encapsulated (Fig. 7), but the main structure of the node had been replaced by cells resembling those of the primary tumor (Figs. 8 and 9).

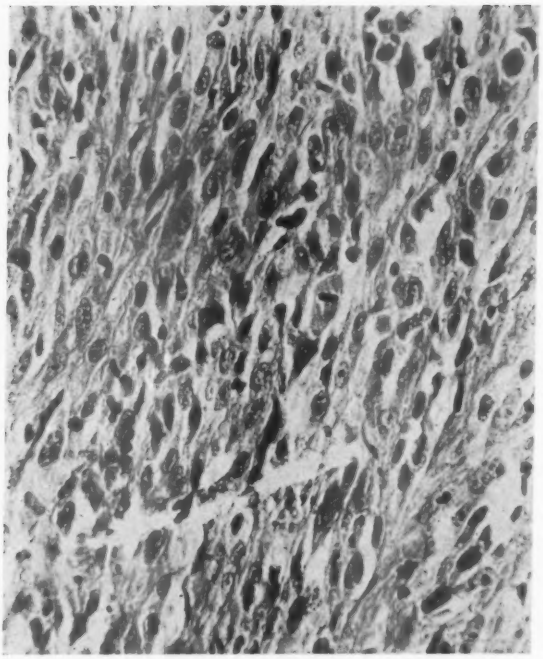

Fig. 8.—Detail of section shown in Fig. 7. (Hematoxylin and eosin stain. Green filter. Magnification, ×550; reduced ¼.)

The patient's postoperative course was good and, apart from the development of a hematoma which required evacuation, healing was uneventful. Eight months later there had been no recurrence or sign of further metastasis.

DISCUSSION

Peripheral fibrosarcoma of the jaws is not a common condition. Bernick[1] states that in his survey of 864 growths of the gingiva and palate this tumor constituted 1.2 per cent of all gingival growths. It should be pointed out, however, that Bernick has included under the term "growths" such conditions as "chronic inflammation (26.1 per cent), scar tissue (11.4 per cent), pyogenic granuloma (5.5 per cent), leukoplakia (4.9 per cent), and granulation tissue (2.8 per cent)." Thoma[2] is of the opinion that the tumor is found more frequently as a peripheral lesion than as a central lesion.

The case described here presents some unusual features:

In the first place, the histology of the main tumor mass, with its many mitotic figures, suggests a tumor of very rapid growth. Clinically, however, this was not so, for the patient reported that it took about a year for the tumor to reach a diameter of approximately 1 inch; therefore, it was growing for at least this length of time. It is interesting to speculate if this slow growth could be the result of the relative avascularity of the mass. This

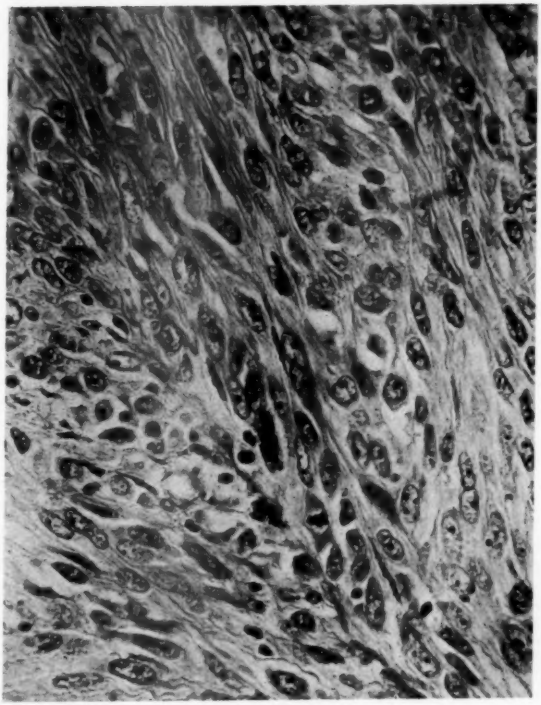

Fig. 9.—Detail of section shown in Fig. 7. (Heidenhain's iron hematoxylin and van Gieson's stains. Magnification, ×600; reduced ¼.)

avascularity might also account for the central zone of necrosis, since the blood supply was insufficient for proper cell nutrition so that as fresh cells formed by division others became necrotic.

Tooth resorption associated with malignant neoplasia is not uncommon, but it is usually associated with multinucleated giant cells. In the present case, however, these cells did not appear to be present in the tissue examined.

The most common route for dissemination of a sarcoma is via the blood stream. In this case, however, the only metastasis found to date has been in a lymph node which "drained" the area of primary growth. This, again, might be a feature related to the poor blood supply of the tumor, there being few vessels with walls sufficiently thin to break down under trauma and allow the neoplastic cells to enter the circulation.

It is customary to think of sarcomas as occurring in children, adolescents, and young adults and to consider their occurrence in patients over 40 years of age as unusual. Several authors, however, in describing tumors of all areas and not just of the jaws, point out that the main incidence is in patients 31 to 35 years of age.[3] Fibrosarcoma of bone may also occur in an even older group of patients. These cases are usually related to predisposing bone lesions,

such as those that occur in Paget's disease. The case described here involved a patient whose age was not far removed from the maximum age usually assigned to fibrosarcoma of bone.

We have knowledge of seven cases of mandibular fibrosarcoma treated surgically by colleagues; five were central and two were peripheral in origin. In five of these cases treatment was apparently successful, since there had been no recurrence after three years. This would seem to differ from the results of treatment of fibrosarcomas elsewhere in the body, where the prognosis seems poor. It may be that the peripheral oral lesions are treated more successfully as a result of earlier diagnosis because of their greater accessibility, or there may be some inherent difference in the lesions themselves.

The case presented here demonstrates that even when, by their clinical history or nature, lesions appear to be slow growing or benign, a detailed examination should be made and a histologic report obtained if there is any doubt as to the diagnosis. In spite of the difficulty of making a definite clinical diagnosis and the risk involved in local surgical interference, some peripheral fibrosarcomas appear to respond well to immediate and thorough surgical removal.

SUMMARY

A case of fibrosarcomatous epulis which showed some unusual features has been described.

It is suggested that mandibular fibrosarcoma may have a better prognosis than fibrosarcomas occurring elsewhere in the body.

The importance of complete investigation is emphasized. In all doubtful cases a histologic examination should be performed.

The authors wish to thank Mr. F. G. Godman of the University of Bristol Medical Photographic Department for the clinical photographs and Mr. B. G. H. Levers of the Dental School for help with the photomicrographs.

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ODONTOGENESIS IMPERFECTA

Report of a Case

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HEREDITARY dental defects may involve either the enamel or the dentine. The literature contains no reports of cases in which both the amelogenesis and the dentinogenesis of teeth may have been affected simultaneously. Therefore, the following case is considered worth reporting.

CASE REPORT

A 7-year-old white girl was first seen on July 24, 1959, at the School of Dentistry, University of Minnesota, for treatment of a hard lump of two weeks' duration in the right cheek. The patient had experienced repeated episodes of swellings in the jaws for about three years. The appearance of these swellings alternated with quiescent periods which usually lasted for one to eight weeks. The mandible was more frequently affected than the maxilla. Two years earlier several teeth had been extracted because they were badly broken down. The child's parents stated that she was slow in erupting her deciduous dentition, which was not completed until she was 4 years old. The previous swellings had usually lasted two to three days and had responded satisfactorily to antibiotic therapy. The parents denied the presence of any such dental defects in their respective families. The father, however, had a

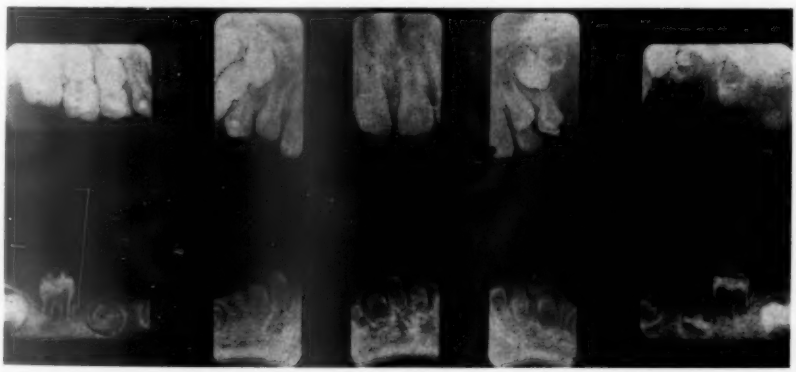

Fig. 1.—Intraoral roentgenograms. Note the anomaly affecting all teeth except in the right upper quadrant. The crowns of teeth are deformed. The enamel and dentine are indistinguishable from each other, and pulp chambers are extremely wide and open.

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vague recollection of some dental anomaly in one of his nephews. There were no apparent environmental factors which could be held responsible for this condition. The patient's two sisters and one brother had normal teeth.

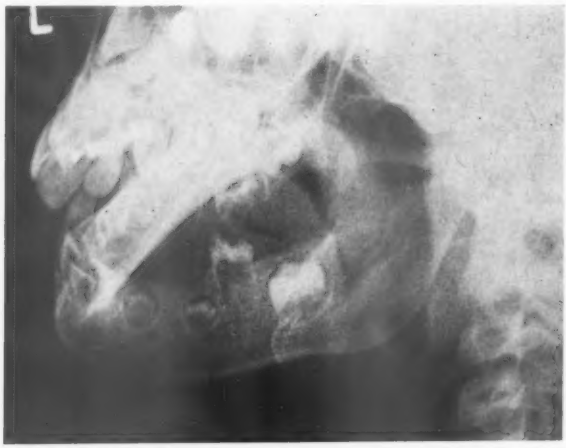

Fig. 2.

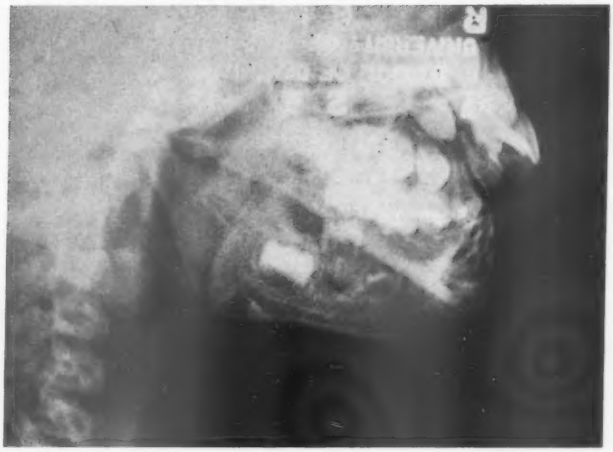

Fig. 3.

Figs. 2 and 3.—Lateral oblique roentgenograms showing lag and defective formation of premolars. Note deficient formation of enamel and dentine, resulting in large pulp chambers and canals. The second permanent molars appear to be normal.

On physical examination, the patient appeared to be a normal child. She was quite active and did not seem to be greatly concerned about the swelling in her cheek. She had a discoloration of the chin, the cheek, and the upper part of the neck, representing a birthmark. Asymmetry of the face, due to the swelling in the right cheek, was apparent. The swelling was firm, brawny, and fixed. It was limited to the posterior part of the cheek in the region of the angle of the mandible. It was not painful, but it was tender on manipulation.

Intraorally, the swelling extended from the first permanent molar to the angle of the mandible. The mucosa was intact, but the periocoronal tissue around the first molar appeared inflamed. On the left side of the mandible there was a similar but smaller swelling with a draining fistula near the crest of the alveolar ridge. The lower first permanent molars were deformed, pigmented, and covered with flecks of brittle enamel. The deciduous teeth in the left upper quadrant presented similar features, but the deciduous and permanent teeth in the right upper quadrant were normal in appearance. Both maxillary permanent central incisors also appeared normal, except for their irregular incisal edges.

Intraoral roentgenograms showed an absence of deciduous teeth in the mandible (Fig. 1). The permanent first molars were partially erupted (Figs. 2 and 3). The unerupted mandibular teeth were abnormal in shape and size and were covered with a very small quantity of indistinguishable enamel and dentine. Root formation was either absent or minimal. The pulp chambers were large and wide open. The posterior teeth in the left upper quadrant also presented similar features. However, the partially formed crowns of the lower second permanent molars and all the teeth in the right upper quadrant appeared normal.

The blood chemistry showed normal serum calcium (10.1 mg. per cent), elevated serum phosphorus (5.1 mg. per cent), and normal serum alkaline phosphatase (14.6

Fig. 4.—Photomicrograph of upper deciduous left cuspid showing degenerative changes in the pulp and formation of denticles.

King-Armstrong units). The hemoglobin was 12.5 per cent and the white blood count was 12,250 per cubic millimeter. The differential blood count was 71 per cent neutrophils, 28 per cent lymphocytes, and 1 per cent monocytes. The bleeding time was 2 and one-half minutes, and the clotting time was four minutes. The urinalysis was within normal range.

The patient was admitted to the hospital on July 28, 1959, and on the following day the lower permanent first molars, the deciduous upper lateral incisor and cuspid, and remnants

of other teeth were extracted. The abscess in the right cheek was drained and yielded more than 7 c.c. of purulent material. The recovery was uneventful, and the patient was discharged from the hospital on the next day. The extracted teeth were fixed in 10 per cent formalin. They were decalcified in Versene to preserve enamel and then were processed in the routine fashion for histologic sections.

Fig. 5.—Photomicrograph of first permanent molar. Note poorly formed enamel and dentine and ill-defined dentinoenamel junction.

The microscopic features were bizarre and atypical. The pulp of the deciduous cuspid showed hydropic degeneration and formation of large spaces by coalescence. This was especially conspicuous in the subodontoblastic layer. The odontoblasts, which were flattened as a result of pressure, were several layers thick in the periapical region. The pulp chamber contained several denticles (Fig. 4). The predentine layer was unusually prominent. The dysplastic changes in the dentine of the first permanent molars consisted of poorly calcified dentine which was lacking in normal architecture or pattern. The dentinal tubules, especially in the peripheral portion, were sparse, irregular, and without any definite arrangement. The dentinoenamel junction was poorly demarcated and lacked scalloping. The enamel showed complete disorganization of its constituents. The enamel prisms were unidentifiable. The enamel seemed to be composed of irregular calcified masses and cellular debris (Fig. 5).

DISCUSSION

This is the first case that we have seen in which there has been a concomitant disturbance in both amelogenesis and dentinogenesis of teeth, thus representing an authentic case of odontogenesis imperfecta. Clinically, the lower first permanent molars presented certain features which were not unlike hereditary enamel dysplasia.[1] The enamel was scant, discolored, and brittle. The crowns of the teeth were deformed and irregular in shape and size. Roentgenographically, the unerupted lower teeth showed root formation to be absent or incomplete like that seen in dentinal dysplasia, except that in the latter condition the crowns of

the teeth are usually normal.[2] Interestingly enough, clinical examination indicated that the teeth of the right upper quadrant were not affected by this malformation. (This observation might not be borne out on microscopic examination of these teeth.) The changes in the pulp were quite impressive. Marked degenerative changes and the presence of denticles in the pulp were quite unusual, especially in such a young patient. The hereditary background of the disease could not be established, but neither could it be ruled out on the basis of the available material. It is conceivable that although the disease had not manifested itself clinically in other members of the family, there might have been microscopic evidence of its existence.[3] Unless more material is available, no definite conclusions can be drawn. Since the eruptive mechanisms are lacking, we doubt that the affected teeth would ever erupt. The treatment of choice, in the future, would be extraction of the teeth and construction of partial upper and full lower dentures for the patient.

SUMMARY

An authentic case of odontogenesis imperfecta in a 7-year-old white girl has been discussed. The cause of the disease is not definitely known. No environmental factors could be linked with the anomaly. Similarly, no genetic background could be established from the limited data available.

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PERIODONTAL CYSTS

An Analysis of Over 500 Cysts

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THIS report is based on more than 500 consecutive dental or periodontal cysts in patients seen at the Glasgow Dental Hospital during a period of eight years.

The Glasgow Dental Hospital is an institution to which patients can be referred by dental or medical practitioners or by other hospitals. As it is situated in an industrial area where dental neglect is common, it might be expected that a large number of patients with a wide variety of unusual oral conditions would be seen.

It should be mentioned that some dental cases are referred to the maxillofacial units and dental outpatient departments of other hospitals in the western region of Scotland. The large number of cysts seen at the Glasgow Dental Hospital does not represent all the cases in the west of Scotland.

METHODS AND MATERIALS

The roentgenograms studied in this series were of those patients who had attended the Glasgow Dental Hospital during a period of eight years. A survey form was drafted on which entries were made under such headings as age, sex, site, tooth/root present, presenting symptoms, etc.

Clinical, operative, and histopathology records were examined to confirm or refute roentgenologic signs.

The roentgenograms of 508 patients were examined. Eleven patients had two cysts and one had three cysts, making a total of 521 cysts.

HISTORY AND PRESENTING SYMPTOMS

Many of the patients came because of a "lump" in the mouth or because the alveolar process had become so enlarged that it was impossible to wear dentures. In some cases a visible deformity of the face had resulted. Others said that there was an unpleasant taste in the mouth.

A minority of patients complained of pain and swelling due to the onset of infection. A remarkable variation in the tolerance and degree of pain was

noted. One patient said that he had discomfort for over seven years and yet had not bothered to take medical or dental advice.

Nine cysts were discovered after a root "disappeared" into the cystic cavity during extraction. One was discovered during routine roentgenography prior to periodontal treatment; another was discovered prior to reduction of an enlarged tuberosity; and in yet another case anesthesia of the lower lip of a few days' duration was the only symptom. In two cases in which a cyst had arisen from a lateral maxillary incisor, these teeth were seen to be excellent examples of dilated composite odontomes of the coronal type.

DURATION

It was found impossible to establish the duration of the cyst in the great majority of cases.

In young patients, however, the maximum duration could be established (age in years less average age in years at completion of root of tooth).

Often teeth had been removed fifteen, twenty, twenty-five, and even fifty years previously. The latter figure was given by two patients who were 66 and 74 years of age, respectively.

SITE

Of the 521 cysts studied, 374 (72 per cent) involved the maxilla and 147 (28 per cent) involved the mandible, a maxilla: mandible ratio of 7:3.

Of the 374 cysts involving the maxilla, 231 (62 per cent) occurred in the incisor region, 27 (7 per cent) in the canine region, 76 (20 per cent) in the premolar region, and 40 (11 per cent) in the molar region. Of the 147 cases involving the mandible, 24 (16 per cent) occurred in the incisor region, 3 (2 per cent) in the canine region, 50 (34 per cent) in the premolar region, and 70 (48 per cent) in the molar region (Table I).

TABLE I. SITE DISTRIBUTION OF 521 CYSTS

SITE	NUMBER	
	MAXILLA	MANDIBLE
Incisor	231	24
Cuspid	27	3
Premolar	76	50
Molar	40	70

SEX INCIDENCE

An average of 63.5 patients presented themselves each year (Table II). There was a total of 271 male patients and 237 female patients, giving a male: female ratio of 1.14:1.0.

It must be remembered, too, that these figures are based on the age when the patient first came for examination and not when the lesion appeared.

AGE ON EXAMINATION

Over the eight years the average age of the male patients was 39 years and that of the female patients was 35 years (Table III). In all but one of the

TABLE II. NUMBER AND SEX DISTRIBUTION OVER EIGHT YEARS

YEAR	MALE	FEMALE	TOTAL
1	17	13	30
2	28	26	54
3	31	35	66
4	23	26	49
5	33	27	60
6	41	40	81
7	51	43	94
8	47	27	74
	271	237	508
	Average: 63.5 patients per year.		

eight years during which a record was kept, female patients came for examination and treatment at a younger age than male patients, in one year by as much as ten years.

TABLE III. AGE AND SEX ON EXAMINATION

YEAR	AVERAGE AGE	
	MALE	FEMALE
1	39	31
2	42	41
3	41	42
4	43	33
5	36	34
6	40	35
7	38	33
8	34	31
	Average 39	35
	Average age over eight-year period: 37	

Three factors may have a bearing on this small difference. First, female patients seek dental attention earlier and more frequently than male patients. Second, as they are more conscious of the value of appearance female patients have any unsightly or unsaveable teeth removed. Third, the maxillary anterior teeth in male patients are more subject to trauma.

Of the 508 patients whose roentgenograms were examined, 275 (54 per cent) had neither teeth nor roots related to the cyst. In the remaining 233 (46 per cent), there were 151 teeth and 82 roots. Only 17 (7.5 per cent) of the combined total of teeth and roots showed any evidence of root treatment (Table IV).

TABLE IV. ANALYSIS OF 508 RADIOGRAPHS WITH FINDINGS REGARDING TEETH, ROOTS, ROOT TREATMENT, ETC.

YEAR	NUMBER OF PATIENTS	ROOT/TEETH ABSENT	TEETH PRESENT	ROOTS PRESENT	NUMBER OF ROOTS TREATED
1	30	19	7	4	—
2	54	35	13	6	4
3	66	45	12	9	2
4	49	29	12	8	1
5	60	36	15	9	1
6	81	35	27	19	2
7	94	39	37	18	3
8	74	37	28	9	4
Total	508	275	151	82	17

Of the 151 teeth, 45 were free of caries, 34 were carious, and 72 had been filled.

AGE DISTRIBUTION

In each decade the number of patients presenting showed a marked pattern (Table V). They increased rapidly to a maximum in the 21 to 30-year-old group and then fell steadily. The youngest patient was 13 years of age and the oldest was 76 years of age. The male patients outnumbered the female patients in all but the groups aged 11 to 20 and 61 to 70 years.

TABLE V. AGE AND SEX DISTRIBUTION, IN DECADES, OF 508 PATIENTS

SEX	AGE (YEARS)						
	11-20	21-30	31-40	41-50	51-60	61-70	70+
Males	23	77	66	48	41	14	3
Females	40	73	41	32	29	19	2
Total	63	150	107	80	70	33	5
Percentage	12.5	30.0	21.0	15.0	14.0	6.5	1.0

COMMENT

The only comparable survey was undertaken at the Mayo Clinic by Stafne and Milhon[1] in 1945. Their study was based on 500 consecutive periodontal cysts occurring in 469 patients, of whom twenty-three had two cysts and four had three cysts. The period covered by the survey is not stated.

The study reported here is based on 521 consecutive periodontal cysts occurring in 508 patients, of whom eleven had two cysts and one had three cysts. The period covered by the survey is eight years.

Stafne and Milhon report that cysts occur more often in the maxilla than in the mandible, the ratio being 3:2. The present series, however, shows, a ratio of 7:3, an increase of 50 per cent for the maxillary cysts. (Table I).

This ratio is explained by a very high percentage of cases in which the maxillary incisor region is affected. Stafne and Milhon state that this region accounts for more than one-third of the maxillary cysts, while the present report shows that about two-thirds (231 out of 374) arise from this area.

The reasons for cysts arising most frequently from this area are as follows: (1) These teeth are retained longer in the dental arch than any other teeth except the mandibular incisors; (2) they are subject to trauma and thus the pulp may undergo painless and unnoticed degeneration; (3) due to the small size of the crown of the lateral incisor, there is a chance of a deep proximal filling causing the death of the pulp; and (4) it should be remembered that globulomaxillary cysts may arise developmentally at the junction of the globular portion of the medial nasal process and the maxillary process.

It is noteworthy that 183 cysts (more than 35 per cent of the total) were found to arise from one tooth, the maxillary lateral incisor.

Cysts are decidedly not a monopoly of age, since only 21.5 per cent were found to occur in patients over 50 years of age.

SUMMARY

1. Over a period of eight years the roentgenograms of 508 patients with cysts were examined. Eleven patients had two cysts and one had three, making a total of 521 cysts. Clinical, operative, and histopathology records were also examined.

2. It was found impossible to establish the duration of these cysts.

3. The maxilla was involved in 374 cysts and the mandible in 147 cysts, a ratio of 7:3. One hundred eighty-three cysts (35 per cent) arose from one tooth, the maxillary lateral incisor.

4. The average age of the patients on examination was 37 years.

5. Thirty per cent of the patients were between 21 and 30 years of age.

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ENDODONTICS

The American Association of Endodontists

Captain Warren J. Hedman, Editor

APPLICATION OF NEWER METHODS TO STUDY THE IMPORTANCE OF ROOT CANAL AND ORAL MICROBIOTA IN ENDODONTICS

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THE practice of employing cultural procedures as sterility tests in endodontic therapy is accepted as being advisable,[1-3] although it is frequently neglected in private dental offices.

A sterile absorbent paper point, moistened with root canal contents, is used to inoculate a tube of culture medium. Meat infusion broths with various enrichments added, as recommended by Grossman in 1938,[4] have been in common use. Prepared as pictured and described in his article, this medium is suitable only for the cultivation of aerobic and air-tolerant bacteria such as staphylococci and streptococci. Leavitt, Naidorf, and Shugaevsky[5] suggested a superior broth culturing method which has come into common use. They pointed out that aerobic as well as strictly anaerobic bacteria can be grown in tubes of deep broth made semifluid by the addition of 0.1 per cent agar to prevent rapid and extensive diffusion of free oxygen into the medium. The presence of extra reducing agents, such as sodium thioglycollate, did not appear to improve the growth of anaerobes, but it may have given the medium an additional reserve, for example, prolonging of shelf life.

In root canals there may be microorganisms that have not been detected by limited cultural procedures. This is realized when highly specialized cultural procedures and phase-contrast and dark-field microscopy are used. The detection of oral treponema in root canals by Mazzarella and associates,[6] Brown and Rudolph,[7] and Hampp[8] is an example.

Perhaps the purpose of microbiologic procedures in endodontics is to enable the operator to feel more at ease when obturating a root canal, and perhaps the

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impression made upon the patient is helpful as well. However, if the purpose of such procedures is to reveal the presence of microorganisms, it would seem that every effort should be exhausted toward their detection. This presumes that the presence of these microorganisms is meaningful.

There is scant evidence that taking cultures has a beneficial influence on the success of endodontic therapy. Rhein and colleagues,[9] in 1928, reported the results of 214 newly treated root canal cases and 278 retreated root canal cases, as judged by follow-up clinical and radiographic examinations. The first group of 71 root canals filled with positive cultures yielded eight failures, while 143 filled with negative cultures, yielded three failures. In the second group 81 root canals filled with positive cultures yielded 15 failures, while 197 filled with negative cultures yielded 17 failures. However, the blood agar medium used to culture the root canal specimens grew only aerobic and air-tolerant organisms, as indicated by their data. Apparently, no direct microscopic examinations were made. By comparison, it is of interest that in the present study of 38 initial root canal specimens in which bacteria were detected, only one-third were positive when cultured on aerobic blood agar. Thus, it is probably true that many more of the culture-negative teeth tabulated by Rhein and associates did contain bacteria when they were filled. On the other hand, it is even possible that only such organisms as can be cultured on aerobic media have the ability to cause endodontic failures, presuming that any can.

Buchbinder[10] reported a 12 per cent failure in ninety-four canals treated between 1930 and 1936 without the benefit of cultures. An 8 per cent failure was realized in 151 canals treated between 1936 and 1940. Canals of the latter group were obturated following two negative aerobic broth cultures. The same sort of criticism applies here as to Rhein's findings. While the results were not actually poor in Buchbinder's findings, in either case, his studies were not run concurrently and the slight improvement in results may have been due to improved clinical technique.

Sommer and his co-workers[3] have discussed types of organisms isolated from root canals and their significance and have summarized the current literature on the subject. According to their summary, and to the best of our knowledge, there is no available information from a controlled study that would indicate which types of microorganism commonly cause postoperative difficulties encountered during the course of root canal treatment or which bacteria, if any, cause failures when left in a well-obturated root canal. Thus, the following questions have been raised from time to time: Why take root canal cultures? What benefit are they to the operator or to the patient? What kinds of organism should excite special concern? What data are there to indicate that certain kinds of organism may be disregarded? What data would indicate that media which grow only aerobic bacteria are adequate? What would indicate that media which grow aerobic and only some anaerobic microorganisms are adequate? The questions regarding kinds of organism assume more importance in view of the fact that when cultures are taken their examination is limited usually to observation of whether or not growth occurs, without regard to what kind of organism may be present.

It was the purpose of this study to investigate some of the fundamental bacteriologic problems involved in endodontic practice; to adapt some more recently developed microscopic and cultural methods to these problems; and to determine, in a pilot study, the feasibility of a larger long-range study. The following are some of the major problems considered: (1) Do relationships exist between the flora of canals at initial instrumentation and the clinical diagnosis of pulp or periapical disease? (2) What organisms present in root canals are most persistent during root canal treatments? (3) What is the relationship between the flora of the root canal and the flora of the oral cavity? (4) What organisms are involved in postoperative disturbances which occur occasionally during the course of root canal treatments? (5) What organisms may represent a potential danger to the patient as well as deter endodontic treatment? (6) When a root canal is treated and obturated without the operator's knowledge of the bacteriologic state of the tooth and the treatment is based only on clinical observations, what organisms persist most commonly and are finally sealed in the root canals? (7) Would the treatment failures that occur relate more to positive bacteriologic findings than to shortcomings in clinical procedures, such as the accidental overfilling of a canal or failure to achieve an apical seal? Findings related to the first three of these problems are discussed here.

MATERIALS AND METHODS

Teeth were classified as being *open* or *closed*. *Open* refers to those teeth whose pulp cavities were open to the oral flora at the time root canal therapy was initiated. *Closed* refers to teeth whose pulp cavities were closed from egress of fluids from the oral cavity at the initial appointment.

Clinical Procedures.—The teeth undergoing treatment were isolated by a rubber dam at each treatment period, and the area of operation was swabbed with a disinfectant.

Usually, two root canal culture specimens were collected from each tooth at each treatment period, including one specimen collected upon entry into the canal at the final treatment period prior to obturation of the canal.

At each treatment, the first culture specimen was collected as soon as the canal was exposed and the medicated dressing removed. Then, when possible, a second culture specimen was taken following instrumentation and chemical cleansing of the canal.

Root canal specimens were collected by means of a sterile paper point which was allowed to remain in the canal for one minute. The point then was transferred to a collecting vial containing 0.5 ml. of 0.1 per cent buffered cysteine solution, as recommended by Howell.[11]

A sampling of the oral flora was obtained at each treatment period. This was done by having the patient rinse his mouth vigorously for one minute with 20 ml. of sterile 1 per cent peptone water.

Bacteriologic Laboratory Procedures.—

Microscopic examinations: Each mouth rinse and root canal specimen was subjected to (1) phase-contact microscopic examination of wet mounts, (2)

dark-field microscopic examination of wet mounts, and (3) bright-field microscopic examination of gram-stained dry smears.

Agar plate cultures: Each mouth rinse and root canal specimen was streaked on plates of three types of agar media: (1) blood agar base (Difco) containing 5 per cent horse blood, incubated anaerobically at 37° C. for forty-eight hours; (2) blood agar base (Difco) containing 10 per cent horse blood and 1 per cent soluble starch (Baker), incubated anaerobically at 37° C. for forty-eight hours; and (3) serum starch agar adapted from Kasai,[12] containing 2 Gm. casitone (Difco), 0.5 Gm. yeast extract (Difco), 0.5 Gm. glucose, 2 Gm. soluble starch (Baker), 0.5 Gm. sodium chloride, 2 Gm. agar, and 5 to 10 ml. horse serum per 100 ml., incubated anaerobically at 37° C. for seven days.

The agar plates for anaerobic incubation were placed in a vacuum desiccator jar equipped with a stopcock. Anaerobiosis was achieved by evacuating and flushing the jars three or more times with natural illuminating gas and absorbing residual oxygen with pyrogallol solution made alkaline with sodium bicarbonate, according to a method adapted from Howell and Pine.[13] Approximately 2 Gm. pyrogallic acid and 4.5 Gm. sodium bicarbonate were placed together on one side of the glass partition in the bottom of an open spray dish. The bottom of the empty half of the spray dish was nearly filled with water from a pipette after the spray dish was placed in the desiccator jar. Each time the flushing process was completed, a moderate negative pressure was left in the jar; the jar was then rocked slightly to spill the water into the side of the spray dish containing the dry reagents. In this way carbon dioxide was liberated for those anaerobic bacteria which are beneficially affected by it.

Each of the anaerobic jars contained a vial of methylene blue solution which indicated anaerobiosis.

Observation and differentiation of colonies of bacteria on agar plates and the picking of individual colonies for study with a short needle were facilitated greatly by the use of a dissecting microscope.

Broth cultures: The paper point remaining in the specimen vial, from each root canal, was placed into a tube of semifluid fluid thioglycollate medium (BBL) which had been enriched with 0.3 Gm. phytone (Difco), 0.05 Gm. cysteine hydrochloride, and 0.1 Gm. soluble starch (Baker) per 100 ml. medium, as adapted from Leavitt and associates and Howell and Pine. The medium was prepared in 15 mm. by 150 mm. screw-cap tubes, filled two-thirds full, and autoclaved in the usual manner. These specimens were incubated at least one week prior to reading by gross visual observation and by microscopic examination with phase-contact microscopy and/or gram-stained smears. Recently, this medium was replaced with thioglycollate medium (BBL) No. 01-135C. Cysteine and soluble starch were added, although this is optional for routine clinic use.

Identification of Microorganisms.—Microorganisms were described and identified according to characteristics described in *Bergey's Manual of Determinative Bacteriology*,[14] with the following additions and modifications:

Microscopic Examination

Miscellaneous cocci—all coccal forms, including those which formed chains.

Rods—restricted arbitrarily to rod forms that had a maximum length of 5 microns. Fusiform bacilli were not included here.

Unbranched filaments—restricted arbitrarily to rod forms in excess of 5 microns in length. These often extended to 50 or more microns in length and ranged from 0.2 to 1.0 micron in diameter.

Large, pointed filaments—1.0 to 1.7 microns in diameter and 5 to 50 microns in length, although most of them were 10 to 20 microns in length. These gram-variable forms were differentiated from the gram-negative, smaller, pointed fusobacteria. Particularly, they were differentiated from *Fusobacterium polymorphum*, which is considered to be 0.2 to 0.5 micron in diameter and 8 to 16 microns in length. While the word *pointed* was used as a general description, some large, pointed filaments that were observed had gently tapering, rounded ends. None of these had swollen, flattened, or concave ends. Many of the shorter forms had a central transverse line of division, and some were slightly pinched at this point.

Branched filaments—only truly branched forms of rods or filaments.

"Racemose" forms—of dubious taxonomy, as are "falciform" microorganisms. Beust[15] pictured them as aggregations of one or more filaments with many coccal or fusiform bodies. These forms are detected more readily from scrapings taken from various regions of the mouth. The "falciform" type was not encountered in this study. Tunnicliff and Jackson[16] suggested that such aggregations may be the normal form of growth of such organisms. Observations that we have made of these forms indicate that they are not true fungi.

Vibrio forms and spirillum forms—small, curved, and s-shaped rods, considered to be members of a single species in the human mouth. Park and Jeynes[17] referred to these as *Selenomonas sputigenum*, whereas MacDonald and co-workers[18] preferred to use the genus *Spirillum* in place of *Selenomonas*.

Culture Identification

Anaerobic streptococci—streptococci isolated anaerobically, which would grow only anaerobically on subculture.

Microaerophilic streptococci—isolated only anaerobically but grew aerobically on subculture.

Typical lactobacilli—short, gram-positive nonspore-forming rods producing typical lactobacillus colonies; grew moderately well in an enriched medium at pH 5.0.

Actinomyces israelii and Actinomyces bovis—identified according to characteristics given in *Bergey's Manual of Determinative Bacteriology*; catalase negative.

Leptotrichia buccalis—resembled actinomyces in structure; catalase positive and grew aerobically, fitting the description given by Howell and his associates.[19]

Bacteroides species—included *Bacteroides melaninogenicus*, which were recognized easily by the production of black colonies on anaerobic blood agar after five or more days, as described by Burdon.[20]

Diphtheroid bacilli—catalase-positive, gram-positive, nonspore-forming pleomorphic rods which did not display true branching; aerobic or microaerophilic.

Neisseria species—of the nonfastidious species; grew on nutrient agar when subcultured.

RESULTS

The laboratory findings obtained by examining the initial specimens from fifty-seven teeth of forty-eight patients are shown in Table I.

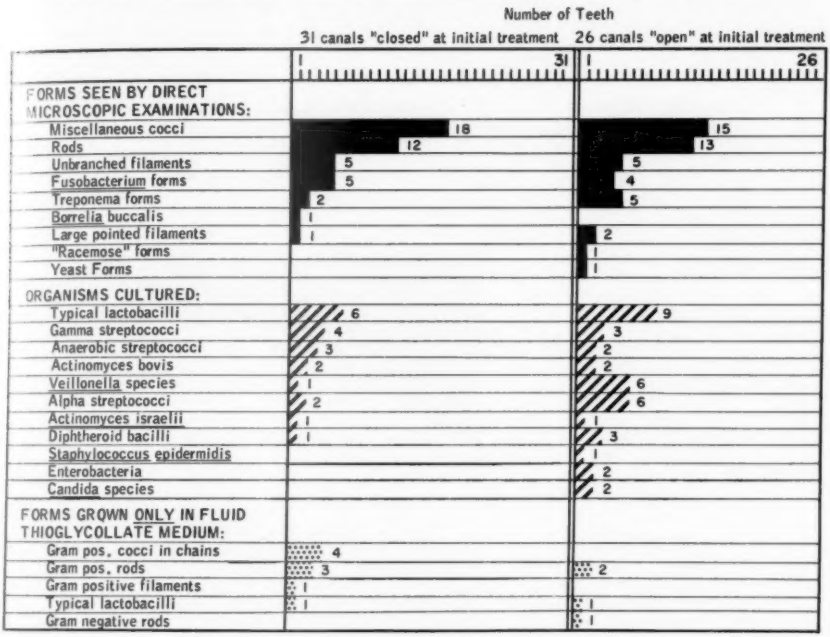
TABLE I

	CLOSED TEETH	OPEN TEETH	TOTAL TEETH
Number of teeth examined	31	26	57
Number with positive findings	22	17	39
Microscopic examinations positive	19	15	34
Cultures on agar medium positive	10	13	23
Cultures in thioglycollate medium positive	16	15	31
Total cultures positive	16	16	32

Only one of the initial specimens that were negative in thioglycollate medium was positive on enriched solid agar, while almost 26 per cent of the total root canal specimens that were positive in thioglycollate broth were negative on agar plates.

Organisms detected in initial specimens that were obtained from open and closed teeth at the first treatment period are listed in Table II. "Racemose" and yeast forms were detected microscopically. *Staphylococcus epidermis*, enterobacteria, and candida species were detected from open canals only. Brown and Rudolph[7] also failed to detect yeasts or many staphylococci in their studies of closed teeth. Contrary to the findings of Brown and Rudolph, but in agreement with those of Winkler and Van Amerongen,[21] lactobacilli were detected in specimens from both closed and open canals in the present study. However, lactobacilli as well as treponema, veillonella, alpha streptococci, and diphtheroids, were detected less frequently in closed-canal specimens than in open-canal specimens. While the predominating organism may appear to be the lactobacillus, if the various types of streptococci are grouped together they are seen to constitute the most common type of organism isolated. Eight out of fifteen positive closed canals and twelve out of sixteen positive open canals yielded streptococci. This agrees with the general findings of other investigators cited. Brown and Rudolph and Hampp did not report finding borrelia in specimens

Table II. Microbiologic Findings in Examinations of Root Canal Specimens



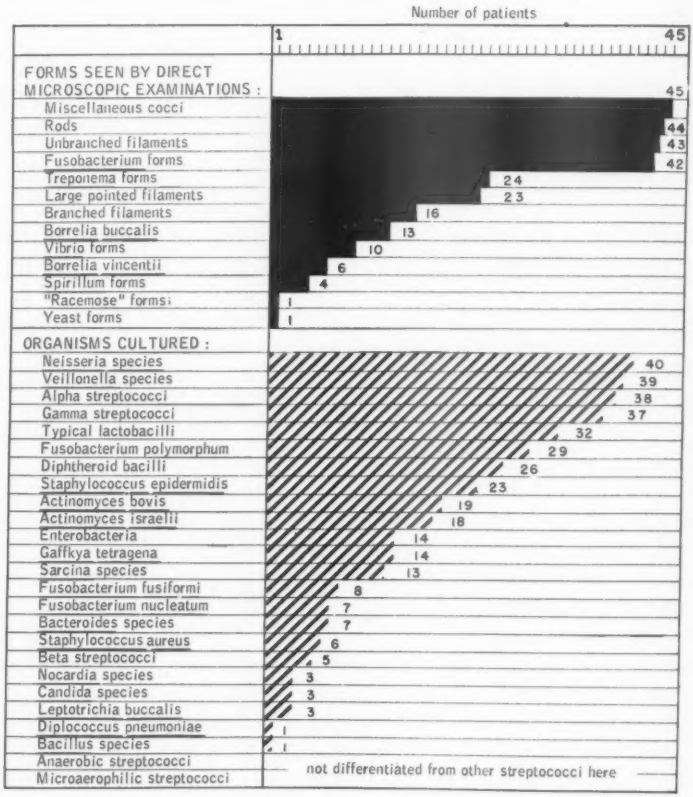
of closed teeth. Borrelia were observed in an initial specimen from a closed pulp cavity in this study. However, this was from a multirooted carious tooth. Trichomonads were detected in one open tooth.

Initial bacteriologic findings were compared with a number of clinical classifications of teeth studied, including various symptoms, radiographic findings, and clinical diagnoses. Generally, there existed a lack of distinguishing correlation between flora and clinical classifications. Because of insufficient data in the various groups, no meaning can be attached to any tendencies observed. One interesting comparison involved six teeth considered to possess periapical granulomas. Not one of these six yielded growth on any of the solid media; yet, three of them yielded growth of gram-positive rods, cocci, and unbranched filaments in the thioglycollate medium only. These results are in contrast to findings in groups with such diseases as pulpitis, necrosis, or gangrene, in which approximately one-half to two-thirds of the initial specimens yielded positive findings on microscopic examination and culture on agar media. Many more such teeth must be studied to substantiate this observation; however, the higher incidence of positive cultures in thioglycollate medium has been pointed out previously in this article.

The types of organisms detected at the beginning and end of five successive treatments are listed in Table III. One of the fifty-two cases required five treatments before clinical symptoms disappeared and canal obturation could be

achieved. A few teeth which were opened to drain because of postoperative complications during treatment were not recorded past the time at which they were opened. Mechanical and chemical cleansing provided a marked depletion of the microbial flora during treatments.

Table III. Total Microbiologic Findings in Examinations of Two or More Mouth Rinse Specimens From Forty-Five Patients



Microscopic examinations revealed that cocci and rods persisted longer. Streptococci and lactobacilli were the most persistent forms detected by cultures, with the exception of a gram-negative rod detected in one case at the end of the fourth treatment. The last three survivors may be attributed to contamination; however, the presence of clinical symptoms prevented obturation of the canals at an earlier appointment. The presence of the organisms may have had a direct bearing on the persistence of clinical symptoms, such as tenderness or an excessively moist canal.

The occurrence of organisms in the total oral flora of forty-five patients is tabulated in Table IV. This was determined from two to five mouth rinses of

each patient; usually three mouth rinses were taken. The method of sampling is reflected in the results. Rosebury and colleagues[22] and Morris[23] used more vigorous methods of obtaining specimens and recovered organisms from restricted areas of the mouth. A higher occurrence of certain adherent branching and filamentous organisms was shown by them. We encountered the same difficulty in cultivating vibrios and filamentous bacteria as was encountered by Brown and Rudolph.

TABLE IV. PERSISTENCE OF MICROBIAL FORMS DURING ROOT CANAL TREATMENTS

TIME SPECIMENS TAKEN DURING TREATMENT	OPEN	CLOSE	OPEN	CLOSE	OPEN	CLOSE	OPEN	CLOSE	OPEN	CLOSE
Treatment	1		2		3		4		5	
Number of teeth sampled at the time	52	49	50	29	16	9	5	2	1	1
Number of sample specimens positive	36	15	13	5	3	0	1	1	1	1
Microscopic findings										
Miscellaneous cocci	30	10	10	2	2					
Rods	23	9	8	3	3					
Unbranched filaments	11	1	4	2						
"Racemose" forms	1	1								
Treponema species	7	1								
Borrelia buccalis	2									
Fusobacterium forms	9		2							
Large, pointed filaments	3									
Yeast forms	1									
Culture findings										
Staphylococcus epidermidis	1									
Alpha streptococci	7		2							1
Gamma streptococci	8									
Anaerobic streptococci	5	2	4						1	
Enterobacteria	2	1								
Veillonella species	7									
Typical lactobacilli	15		3							
Actinomyces israelii	2									
Actinomyces bovis	4									
Diphtheroid bacilli	4		1							
Candida species	2									
Forms Grown Only in Thioglycollate Medium										
Gram-positive cocci			1							
Gram-positive cocci in chains	4		1							
Gram-positive rods	5	4	1	1						
Gram-negative rods	1							1		
Gram-positive filaments	1									
Typical lactobacilli	2									
Gamma streptococci		1								
Fusobacterium polymorphum		1								

Certain relationships between the total oral flora and that of a restricted area in the mouth, such as the root canal may be pointed out. That the over-all flora of the mouths and the flora of the root canals are not identical may be seen

by comparing Table II with Table III. The flora of the open canals tends to approach that of the mouth more than that of the closed canals; however, this would be expected. *Staphylococcus epidermidis, Staphylococcus aureus,* vibrio forms, spirillum forms, *Gaffkya tetragena,* sarcina, beta hemolytic streptococci, nocardia, leptotrichia, *Diplococcus pneumoniae,* and bacillus species present in the mouths of some patients were not detected in root canals of the same patients. Neisseria, which was detected in the mouths of forty of the forty-five patients, was not detected in their root canals.

When all of the streptococci were considered as a group, they constituted the organisms detected most often in the oral flora. All mouthwash specimens contained either alpha or gamma hemolytic strains of these organisms. Morris also found that these were the most numerous organisms seen in the individual oral samplings.

Staphylococcus aureus was detected in the mouths of six of forty-five patients. Knighton[24] demonstrated that *Staphylococcus aureus* was a predominating organism in the oral and nasal flora. Leavitt, Naidorf, and Shugaevsky[25] recovered *Staphylococcus aureus* from 13 per cent of 154 root canals. This exceeds the findings of Winkler and Van Amerongen, and Brown and Rudolph; yet none of these studies, including this present one, suggests that *Staphylococcus aureus* is the principal organism recovered from root canals. The low incidence of beta hemolytic streptococci, *Streptococcus pyogenes,* is even more obvious.

The bacteroides detected in the mouths of approximately 35 per cent of the patients were almost exclusively *Bacteroides melaninogenicus.* None were detected in root canal specimens.

Tables II, III, and IV give no indication of the order of predominance of the different types of microorganisms within the specimens in which they were detected, but they do indicate the number of root canals or mouths from which the various microorganisms were obtained. A tabulation could have been made of the averages of absolute counts of various organisms detected, but that was beyond the scope of this project. Instead, differential counts were made to determine the order of predominance of one form over another during the initial microscopic examination of all specimens. This order was recorded as one, two, three, etc., indicating most prevalent, next most prevalent and so on for each microbial form detected in each specimen. An arithmetic mean ($\overline{X}$) was obtained to express the average order of predominance of each form in all specimens in which each was observed. An index was expressed as the reciprocal of $\overline{X}$ multiplied by 100, that is, $\frac{100}{\overline{X}}$. This provides an index of the average microscopic predominance of the different forms by which they can be compared.

Similar indices were calculated for culture findings on agar plates, although it should be borne in mind that the degree of growth on artificial media is influenced by more factors than just the numerical occurrence of the organisms. Here, growth of an organism was tabulated approximately as 0.5 (1 to 5 colonies), 1.0 (5 to 50 colonies), 2.0 (50 to 200 colonies), and 3.0 (profuse growth). Thus, a semiquantitative estimate of growth was made for the organisms cultured

from each specimen. A mean ($\overline{X}$) was calculated from the numbers recorded for each of the organisms in all specimens from which they were cultivated. For comparison, a cultured index was expressed as 100 $\overline{X^2}$, indicating the average degree of growth attained by the various organisms on agar plates. It was in this manner that indices for microscopic and culture methods were prepared on a scale of 1 to 100, arbitrarily chosen for the organisms detected in seventy-one mouth rinses from twenty-seven of the patients and initial specimens from twenty-six open and thirty-one closed teeth, as tabulated in Table V.

TABLE V. RELATIVE PREDOMINANCE OF ORGANISMS IN THE SPECIMENS IN WHICH THEY WERE FOUND

TOTAL ORAL FLORA		FLORA OF CLOSED TEETH		FLORA OF OPEN TEETH	
Microscopic Examinations		*Microscopic Examinations*		*Microscopic Examinations*	
Miscellaneous cocci	100	Miscellaneous cocci	97	Miscellaneous cocci	82
Rods	46	Rods	65	Rods	62
Unbranched filaments	34	Treponema forms	40	Yeast forms	33
Fusobacterium forms	28	Fusobacterium forms	38	Treponema forms	28
Large, pointed filaments	26	Borrelia forms	33	Fusobacterium forms	27
Branched filaments	25	Unbranched filaments	32	Unbranched filaments	25
Vibrio forms	25	Large, pointed filaments	17	"Racemose" forms	25
Borrelia species	25			Large, pointed filaments	22
"Racemose" forms	22				
Spirillum forms	21				
Yeast forms	14				
Culture Examinations		*Culture Examinations*		*Culture Examinations*	
Alpha streptococci	73	Alpha streptococci	63	Anaerobic streptococci	90
Actinomyces israelii	66	Anaerobic streptococci	54	Enterobacteria	90
Neisseria species	63	Typical lactobacilli	46	Gamma streptococci	70
Gamma streptococci	55	Gamma streptococci	40	Alpha streptococci	46
Veillonella species	48	*Actinomyces bovis*	40	Veillonella species	40
Gaffkya tetragena	40	Veillonella species	10	Diphtheroid bacilli	40
Nocardia species	40	*Actinomyces israelii*	10	Typical lactobacilli	35
Bacteroides species	33	Diphtheroid bacilli	3	*Actinomyces israelii*	10
Staphylococcus epidermidis	32			*Staphylococcus epidermidis*	10
Fusobacterium species	32			Candida species	5
Typical lactobacilli	29				
Sarcina species	24				
Beta streptococci	24				
Staphylococcus aureus	20				
Enterobacteria	17				
Diplococcus pneumoniae	10				
Leptotrichia buccalis	10				
Bacillus species	10				
Candida species	7				

DISCUSSION

The limitations of using anaerobic chambers for cultures in general endodontic practice may be justly emphasized. Tubes of deep semifluid broth, when used correctly, are capable of growing strictly anaerobic as well as aerobic and intermediate microorganisms, as indicated by Leavitt and associates. Yet we found that even such a medium was not always capable of growing organisms which were detected microscopically. As reported by Brown and Rudolph, even the stained smears recommended by Grossman in 1938 may surpass such culture procedures.

The positive results obtained here consistently lagged slightly less than 30 per cent below those reported by Brown and Rudolph, who detected organisms microscopically in 90 per cent of seventy closed teeth. Approximately 71 per cent of the seventy teeth yielded positive cultures anaerobically on blood agar, and 85 per cent of twenty-six specimens tested were positive in thioglycollate broth. It is possible that the teeth studied here contained fewer organisms or that more material was removed and discarded from the pulp chambers before initial specimens were collected. The fair degree of consistency with which results in our study were below theirs would indicate that none of the various methods of analysis were at fault.

Results obtained in open and closed canals do not agree with those of a larger study by Winkler and Van Amerongen, who used a single semifluid culture medium. They obtained positive initial cultures from 47 per cent of 733 teeth, but of their 471 vital pulps, 192 yielded positive cultures whereas necrotic pulps yielded 39 positive cultures.

Here again the question arises as to how much material was removed and discarded from the pulp chambers before the initial cultures were taken. Study of a larger number of teeth, together with sampling of pulps at various depths, is indicated.

The apparent superiority of thioglycollate broth over the solid media used in this study and the study reported by Brown and Rudolph may be due to the rapidity of placing the inoculum into an environment of low oxidation-reduction potential in the broth. Since thioglycollate broth was the only medium to produce growth from the teeth associated with granulomatous lesions of long duration, the study of more such teeth to verify the observation and to learn more about the ability of this semifluid medium to overcome antibacterial tissue factors would be of interest.

Forms observed microscopically which could not be cultured appeared to resemble the more commonly occurring forms, such as gram-positive cocci and rods, rather than such unusual forms as the more fastidious treponema and Borrelia. Actually, in all specimens in which spirochetes were observed, they were accompanied by one or more other bacterial forms which were cultured readily. This suggests that, for some reason, conditions were not always suitable for obtaining growth of the more commonly encountered cocci and rod forms or that, for some other reason, depending on their state of viability or their nutritional environmental requirements, they differed from the cultivable forms which they resembled.

The fact that lactobacilli were detected so readily in root canal specimens may have been influenced by their tendency to grow well on the starch agar medium that was used in this study. Lactobacillus growth on starch agar sometimes exceeded the growth on lactobacillus selective medium (BBL) when parallel inoculations, incubated anaerobically, were compared.

In the studies of oral flora and sputum in this laboratory, as well as the studies by Burdon, it is suggested that *Bacteroides melaninogenicus* is a common inhabitant of the mouth and related areas. Because of its lack of distinctive

colony characteristics following short incubation and because of its tendency to grow best in mixed cultures, this microorganism is easily overlooked. The following simple method was adapted from Burdon for application to the study of root canal flora. Blood agar slants were inoculated and the tubes were affixed with pyrogallol seals, as described by Howell and Pine. Dry pyrogallic acid and sodium bicarbonate was substituted for aqueous solutions, and 0.5 ml. of water was added to the cotton before the tube was sealed with a rubber stopper. Five to ten days of incubation revealed the characteristic shiny black *Bacteroides melaninogenicus* colonies accompanied by leaching of hemoglobin from the agar surounding the colonies. Thus, identification could be realized without the need of opening the culture tube and liberating the foul odor associated with such cultures.

Microscopically detected cocci and non-beta hemolytic streptococci were the forms most commonly detected in root canal specimens and in the oral flora. They also predominated over other forms within individual specimens, as was found by Morris. Anaerobic streptococci were well represented in root canals. The low occurrence of *Streptococcus pyogenes* and *Staphylococcus aureus* does not suggest that these more classic pathogens were responsible for the pulp damage commonly observed. With the dubious exception of a borrelia in one closed tooth, it is also of interest that closed canals contained a slightly restricted representation of the open canal flora and that open canals contained a more restricted representation of the total oral flora, as though only some of the oral flora can survive in a root canal. Thus, the movement of bacteria into the root canal does not seem to typify an aggressive invasion. Rather, there seems to be more of a passive migration restricted by degree of exposure, environment within the canal, and perhaps viability of the pulp. This possibility is supported by Rhein's inability to experimentally infect pulps in dogs with streptococci and oral flora. Thus, there arises the question: Are the more commonly recovered root canal bacteria responsible for initiating the destruction of vital pulps? Or do they only secondarily invade injured or moribund pulps?

The fact that more classic pathogenic bacteria, such as *Streptococcus pyogenes* and *Staphylococcus aureus,* are recovered from root canals on occasion suggests a need for the endodontist to obtain knowledge of the kinds of organism present in the root canal being treated. In this way he may deal more judiciously with the patient as well as with the tooth.

Some further improvements suggested by this study are being considered:

1. Removal of more than one initial sample from root canals at various depths.

2. The use of duplicate specimens—one to be placed in 1 c.c. of semifluid reducing medium in a slender vial to minimize exposure to free oxygen during transportation and the other to be placed in a similar vial of filtered broth which may be centrifuged at high speeds. Centrifugal action would concentrate organisms that are freed from the paper point for microscopic examination.

3. Comparison of the anaerobic "fusiform agar" medium used by Richardson and Jones[26] for cultivation of filaments with the anaerobic blood agar medium used here.

4. A survey of root canals for bacteroides, as described.

5. Exploration of the feasibility of quantitative microscopic counts of root canal flora.

SUMMARY

1. Root canal specimens from fifty-seven teeth and mouth rinse specimens from forty-five patients were examined directly by phase-contrast, dark-field, and bright-field microscopy and by aerobic and anaerobic culture on agar media. Deep semifluid broth was also inoculated with root canal specimens. It was reaffirmed that microorganisms could be detected microscopically which could not be detected by cultures alone.

2. The flora of open and closed teeth were compared. That of the latter was found to be a similar though restricted representation of the former. Non-beta hemolytic streptococci were found to predominate in both. Noncultivable spirochetes were found only with other mixed cultivable flora. Forms observed in root canal specimens which yielded no growth resembled more commonly observed forms, such as gram-positive cocci and rods.

3. Root canal flora tabulated in relation to endodontic treatments diminished from one treatment to the next, as well as from the beginning to the end of individual treatments.

4. When root canal flora were compared with clinical diagnoses, organisms were detected by all methods and to about the same extent as in all canals, regardless of diagnosis, with the possible exception of teeth with granulomatous involvement. These organisms were detected only by microscopic examination and semifluid broth cultures.

5. Root canal flora and total oral flora of the patients were compared. They differed in a marked restriction of some oral forms and species from the root canals. Beta hemolytic streptococci and *Staphylococcus aureus* found in mouth rinses were not found in root canals.

6. Indices representing predominance of forms as seen by direct microscopic examination of the oral and root canal flora and the growth of microorganisms on agar plates were recorded for comparison. Coccal forms and non-beta hemolytic streptococci were the most numerous as well as the most commonly detected microorganisms in all flora.

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RESEARCH

A MODIFIED METHOD FOR THE COLLECTION OF HUMAN SUBMAXILLARY AND SUBLINGUAL SALIVA

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THE secretions from the three major salivary glands (parotid, submaxillary, and sublingual) have been studied since early in the twentieth century. Several methods[1-3] for the collection of parotid saliva are available and may be employed with both gustatory and masticatory stimuli. An apparatus for the collection of mandibular saliva, the mixed secretion from the submaxillary and sublingual glands, was first fabricated by Pickerill.[1] This appliance could be used with gustatory and masticatory stimuli, but it did not permit separation of the fluids produced by the different glands. Schneyer[4] later devised a method by which pure submaxillary and sublingual saliva can be obtained. This latter apparatus is limited however, in that masticatory stimuli cannot be used. The present article describes procedures for modifying this appliance so that the separate submaxillary and sublingual secretions can be collected when masticatory as well as gustatory stimuli are used.

METHODS AND MATERIALS

The finished appliance covers the floor of the mouth from the lingual aspect of the anterior teeth to the area of the second molars. The sublingual surface of the appliance has three collection chambers—one anterior and two posterior or lateral. The anterior chamber overlies the opening of Wharton's ducts, and each of the others covers one of the groups of sublingual ducts of Rivinus and Bartholin which lie lateral to the root of the tongue. A plastic tubule leads from each chamber to a collection receptacle outside the mouth (Figs. 1 and 2).

Fabrication Procedure.—First, a primary alginate impression is taken of the lower denture and the floor of the mouth. An excess of alginate about the lingual border of the tray is used to ensure good detail of the sublingual area.

Next, a model is prepared with plaster and is used to make a tray with double-thick baseplate. The tray is extended to within ⅛ inch of the floor

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of the mouth to permit an adequate impression thereof. Perforations hold the alginate in place.

A second impression is taken, with excess alginate about the lingual border of the tray, and a model is poured in stone. This model is surveyed, and all lingual undercuts are blocked out in wax. An 0.001 inch undercut is then

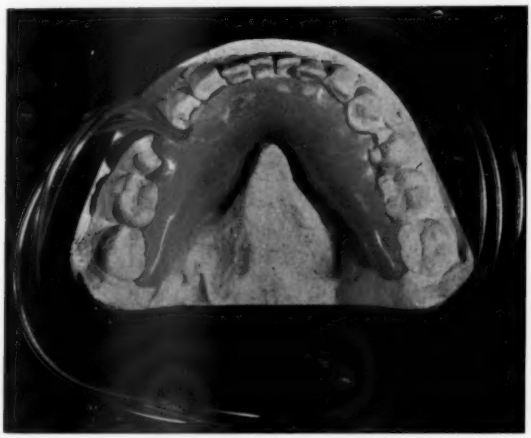

Fig. 1.

Fig. 2.

relieved from the wax along the lingual surfaces of the premolars and molars, gingival to the survey line, to provide retention. The model is now duplicated in stone.

Peripheral seals about the submaxillary ducts (one) and each of the groups of sublingual ducts (two) are prepared by circumscribing lines on the stone

model. Each collection chamber is delineated by a line passing $\frac{1}{16}$ to $\frac{1}{8}$ inch around the group of duct orifices. The anterior limits of the two sublingual chambers and the distal limits of the submaxillary chamber are usually combined. The lines circumscribing these areas are 1 mm. wide and are relieved to a depth of about 1 mm. The model is placed in boiling water for a few minutes and then painted with separating medium, and allowed to cool.

Cold cure acrylic, applied by the "salt-shaker" method, is used for building the appliance. Polyethylene collection tubules, $\frac{1}{16}$ inch (inner diameter), are used to transport the secreted fluid to the exterior. On each tubule the end which is placed in the model should be sealed with soft wax to prevent entrance of acrylic. The collection tubules are run through spaces created by missing teeth or over the anterior teeth. If the tubules are run over the anterior teeth, the appliance must be extended onto the occlusal surfaces of the posterior teeth to form a bite block. This bite block is built up sufficiently to provide clearance for the tubules as they pass between the opposing anterior teeth. The appliance should engage infrabulge areas to hold it against the tissues, and it should engage suprabulge areas of the posterior teeth to prevent applied pressures from forcing it into the soft tissues. Where adequate undercuts are not available on the lingual surface of the teeth, wire clasps or other devices may be used to hold the appliance in place.

After the acrylic sets, the appliance is removed from the model and the chambers above the ducts are cut out with an acrylic stone or a large rose burr. This is done to the inside limits of the peripheral dam and to a depth of $\frac{1}{4}$ inch or more, depending on the size of the papillae of the ducts. The tubule ends, now exposed, are cut and freed of the wax seal. Excess acrylic is ground off the body of the appliance to produce minimal displacement of tissues. The final product is polished to prevent irritation.

The apparatus is placed in the patient's mouth and checked for proper seating and comfort. After the patient has become accustomed to the appliance and the secretion rate is stabilized under the desired test conditions, saliva may be collected for analysis.

When not in use, the appliance is kept in a 0.1 per cent solution of Zephiran hydrochloride.

RESULTS

Functional Efficiency and Effect of Occlusal Bite Plane.—Functional efficiency of the appliance required the collection of submaxillary and sublingual secretions without loss, intermixing, or contamination from an exogenous source (parotid saliva). Several interfering factors had to be eliminated before it was possible to collect saliva when masticatory stimuli were used. The major difficulties encountered were (1) excessive movement of the tongue during mastication and deglutition; (2) insufficient stability of the fitted appliance; and (3) inadequate peripheral seals surrounding the collection chambers, permitting loss of contact between the apparatus and the sublingual mucosa during mastication.

The following procedure was devised to evaluate the efficacy of the fitted appliance. The participating persons rinsed their mouths thoroughly with tap water, and 6 ml. of whole saliva was collected. (One chlorophyll gum tablet* was used as the stimulus.) The test subjects again rinsed their mouths and the appliance was placed in position. A new gum tablet was introduced and 6 ml. of submaxillary saliva was collected. The dye contained in the gum preparation imparted a green color to the saliva. Appliance leakage was detected by the presence of a green color in the submaxillary and/or sublingual fluids. Optical density of the saliva samples was assayed in a Klett-Summerson colorimeter. A red (660 mμ) filter was employed to give relative values of color intensity, while a green (540 mμ) filter was used to measure relative turbidity arising from mucin and debris. Table I presents the results obtained. Persons whose submaxillary secretion had an optical density greater than 6 per cent of that exhibited by whole saliva were eliminated from the study.

TABLE I. EVALUATION OF APPLIANCE FUNCTIONAL EFFICIENCY

SUBJECT NUMBER	OPTICAL DENSITY READING				PER CENT DISCOLORATION	PER CENT TURBIDITY
	660 Mμ FILTER		540 Mμ FILTER			
	SUBMAXILLARY SALIVA	WHOLE SALIVA	SUBMAXILLARY SALIVA	WHOLE SALIVA		
1	18	345	18	435	5.0	4.0
2	1	218	3	310	0.4	1.0
3	7	370	5	475	2.0	1.0
4	18	375	25	400	4.9	6.2
5	13	540	20	640	2.4	3.1
6	4	510	9	625	0.8	1.6
7	6	375	18	440	1.6	4.1
8	5	340	12	400	1.5	3.0
9	2	425	4	490	0.8	0.8
10	194	475	260	600	40.0	43.3

The effect of open-bite versus normal occlusion was determined by processing two sets of appliances—one for normal occlusion and a second with an occlusal bite plane—for each of five subjects. After the desired appliance was positioned, 10 ml. of submaxillary saliva was collected. The stimulus employed was 3.0 Gm. of sugar-sweetened chicle.* The bolus was discarded and a new portion of gum introduced every five minutes. This controlled the inherent variability normally produced by the sweetened chicle and permitted a fairly constant rate of secretion. The mean secretion rate (ml. per min. per gland pair) was calculated by recording the time required for the secretion of a standard 10.0 ml. volume.

An early morning (fasting) saliva sample was obtained with both appliances. A fifteen-minute rest period was allowed between samples. The subjects were then given breakfast and the collection procedure was repeated. The order of application of the two appliances was randomized, and samples were collected for three successive days.

*Supplied through the courtesy of the American Chicle Company, Long Island City, New York.

The results are presented in Table II. The data indicate that the secretion rate for the two appliances did not differ significantly. Similarly, eating did not appear to affect the rate of secretion.

TABLE II. EFFECT OF OCCLUSAL BITE PLANE ON SUBMAXILLARY GLAND SECRETION RATE*

DAY	CONDITIONS OF COLLECTION	ML./MIN./GLAND PAIR	
		NORMAL OCCLUSION	OCCLUSAL BITE PLANE
First	Before eating	1.08	1.08
	After eating	1.12	1.05
Second	Before eating	1.15	1.42
	After eating	1.02	1.19
Third	Before eating	1.22	1.34
	After eating	1.49	1.31
Average rate for 3 days	Before eating	1.19	1.28
	After eating	1.24	1.18

*Data based on mean secretion rates of five subjects.

DISCUSSION

Anatomic evaluation of the submaxillary and sublingual duct systems has indicated that certain variations and anastamoses exist. Jackson and Blount[5] have remarked that the ducts from the sublingual gland, five to fifteen in number, usually open on the minute papillae present along the crest of the sublingual fold. However, the anterior portion of the gland was often found to form a major duct (Bartholin's) which opened on the sublingual caruncula, alongside the submaxillary duct. Sicher[6] states that the anterior or larger portion of the sublingual gland secretes through Bartholin's duct and that this duct either unites with the submaxillary duct or opens close to it. In support of Sicher's observation, Lyon[7] noted that eleven of twenty-two cases studied showed a communication between the sublingual and submaxillary gland duct systems. Conversely, Drennan[8] found that all the ducts of the sublingual gland, as a rule, open separately on the sublingual fold.

Thus, it is extremely difficult to ensure complete isolation of the sublingual and submaxillary secretions, even when the submaxillary duct is directly cannulated. Fortunately, it has been observed[9] that the sublingual glands produce only 2.8 per cent of the total salivary secretion, as compared to 63 per cent for the submaxillary glands. Therefore, if an appliance such as that described here is checked for accurate seal, it should permit collection of relatively pure submaxillary saliva. In addition, there should be a minimal intermixing of the submaxillary and sublingual secretions and only minimal contamination by the parotid secretion.

SUMMARY

An improved method is described for the fabrication of an apparatus capable of collecting submaxillary and sublingual salivas with masticatory as well as gustatory stimuli. Evaluation of the functional efficacy indicated that (1) if the peripheral seals surrounding the collection chambers are adequate and (2) if the appliance has proper retention, then, except for naturally

occurring anastomosis, there will be minimal intermixing of the submaxillary and sublingual secretions and little or no contamination by parotid saliva.

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THE EFFECT OF CALCIUM HYDROXIDE ON DEEP CARIOUS LESIONS

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CONSERVATION of the exposed dental pulp is one of the everyday problems that have beset the general practitioner for decades. The tortuous, ribbonlike canals of the deciduous tooth and the bulbous apical region of the young permanent tooth have proved to be especially difficult to treat from an endodontic standpoint. Alternate methods, such as pulpotomy and pulp capping, have been suggested for these special situations, and there have been varying reports of clinical success.[1-3] In most cases, prolonging the vitality of deciduous or young permanent teeth would be of immeasurable value in the light of future treatment.

Sowden[4] reported on a technique in which only a portion of a deep carious lesion was excavated and a dressing of calcium hydroxide was placed over the remaining portion for the purpose of arresting the existing caries and stimulating favorable dentine changes or "remineralization." It was the intent of this investigation to study the effect of calcium hydroxide in deep carious lesions *where pulpal exposure was highly probable but not actually visible in the roentgenogram.* Such cases are often seen clinically, especially in the rampant caries exhibited in children during the period of the deciduous and mixed dentitions. Excavation of such carious lesions frequently results in exposure, since it is often difficult to distinguish between the remaining carious dentine and the relatively immature but sound dentine. If a treatment method which would classify the status of these borderline cases were available, it would be of great usefulness in operative dentistry. If successful, such a technique would result after a few months in a clearly recognizable, sound cavity floor upon which a suitable restoration could be placed. If pulp exposure was found after the treatment period, it would be still possible to resort to pulp amputation or other accepted methods of therapy.

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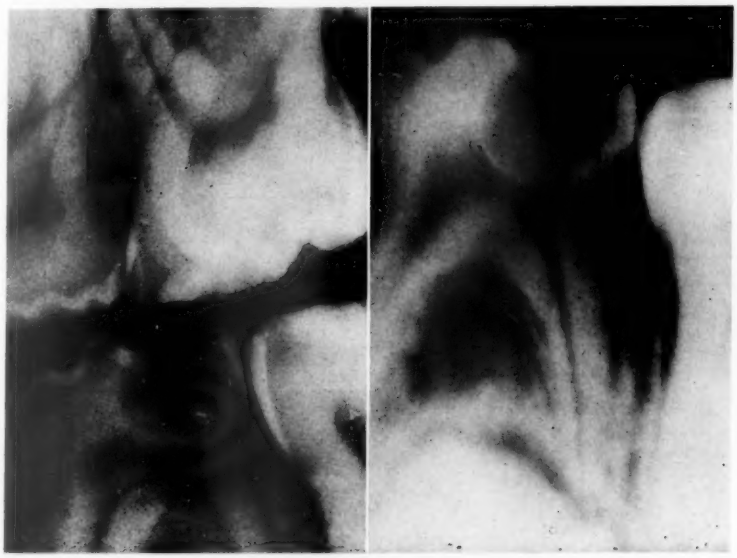
MATERIAL AND METHOD

All patients in the present study were treated in the Pedodontic Department at the University of Washington School of Dentistry. Sixty-six deciduous and young permanent teeth were operated upon and subsequently followed for periods of time varying from seven days to twenty-eight months.

Periapical and bitewing surveys of the teeth were taken prior to surgical intervention, and the presence of a deep carious lesion, closely encroaching upon the pulp but not actually into it so far as could be determined by the roentgenogram, was assured (Fig. 1). Next, 2 c.c. Xylocaine hydrochloride with epinephrine 1:50,000 was administered, and a rubber-dam was placed in position.



A. *B.*

Fig. 1.—*A*, Bitewing roentgenogram of mandibular left first deciduous molar showing a deep carious lesion.

B, Periapical roentgenogram of same tooth. Note nearly indistinguishable radiopaque area occlusal to dental pulp.

Rotary instruments were used to gain access to all decayed areas. A small amount of calcium hydroxide was moistened with sterile water until a heavy paste formed, and this material was deposited on the carious dentine with an amalgam carrier. The calcium hydroxide was then dried, forming a reasonably hard layer 1 to 2 mm. in thickness. If necessary, a matrix band was placed. The rest of the cavity was filled with a soft amalgam mixture and condensed with light pressure (Fig. 2). Periapical and bitewing roentgenograms were taken again immediately following treatment and at periodic intervals thereafter for several weeks. As early as seven days following treatment, a radiopaque area formed on the pulpal side of the residual caries (Fig. 3). This area was evident in nearly every case.

Fig. 2.

Fig. 3.

Fig. 2.—Periapical view of mandibular left first deciduous molar immediately after partial excavation of dental caries and placement of calcium hydroxide paste covered with amalgam restoration.

Fig. 3.—Periapical view of mandibular left first deciduous molar twelve days after placement of calcium hydroxide. Note distinct radiopaque area on pulpal side of residual caries.

Fig. 4.—Periapical view of lower left first deciduous molar six months after treatment. Note thickened radiopaque area pulpal to residual caries beneath amalgam restoration.

Six months later the patients were recalled. Clinical and roentgenographic (Fig. 4) examinations were made, the teeth were anesthetized, and a rubber-dam was placed. The amalgam was removed by means of rotary instruments. The calcium hydroxide and the remaining caries were excavated with hand instruments. The operation was declared successful if the clinical findings were normal, if a firm layer of dentine was found at the base of the cavity after complete excavation of caries, if no pulp exposure was encountered, and if the roentgenogram demonstrated no pathologic condition in the tooth or surrounding tissues. If the procedure was successful, the cavity preparation was completed and a zinc oxide and eugenol cement base was placed. An amalgam restoration was placed in the usual manner. If all criteria for a successful case were evident except for the presence of a pulp exposure upon excavation of the caries, a partial pulpotomy or a vital pulpotomy, depending upon the size of the exposure, was performed.

Twelve months and twenty-four months after the original treatment, the patients were recalled, the teeth were clinically examined, and periapical and bitewing roentgenograms were taken. The treatment was then considered successful if clinical and roentgenographic findings were still negative.

RESULTS

Six months after the initial calcium hydroxide treatment, twenty-seven of the original thirty patients returned for final excavation of the cavities. Fifty-seven of the original sixty-six teeth were re-entered. One tooth had been previously extracted. Those patients who were not available for re-examination were dropped from the study. According to the criterion that treatment was successful if no pulp exposure was encountered on excavation and if the roentgenographic evidence was negative, forty-four teeth were considered satisfactory.

One year and two years following the initial treatment all patients were recalled. Clinical and roentgenographic examinations revealed no failures among cases in which treatment had been previously considered successful. It was thus determined after two years of observation that this technique had succeeded in restoring forty-four out of fifty-seven questionable teeth to a healthy, functional state. This represents a 76 per cent success.

The left-hand column of Table I illustrates that, of the fifty-eight teeth studied, thirty-eight were deciduous teeth and twenty were permanent teeth.

TABLE I

NUMBER OF TEETH IN STUDY			SUCCESS OF TREATMENT AFTER TWO YEARS
Deciduous		38	
Maxillary	13		9
Mandibular	25		19
Permanent		20	
Maxillary	8		8
Mandibular	12		8
Totals		58	44
Per cent success			76

Fig. 5.—*A* and *B*, Bitewing and periapical roentgenograms of lower left first permanent molar showing extensive dental caries.

C and *D*, Same tooth one year after treatment with calcium hydroxide and residual caries. Note radiopaque area pulpal to residual caries. This tooth was considered clinically and radiographically sound.

Of the deciduous teeth treated, thirteen were maxillary and twenty-five were mandibular. Of the permanent teeth treated, eight were maxillary and twelve were mandibular.

The success of this treatment over a two-year period is shown in the right-hand column of Table I. Nine maxillary deciduous teeth and nineteen mandibular deciduous teeth were clinically and roentgenographically satisfactory; thus, in 74 per cent of the thirty-eight deciduous teeth treatment was successful. In eight maxillary and eight mandibular permanent teeth, treatment was successful; thus 80 per cent of all twenty permanent teeth were considered successfully treated.

Fig. 6.—Mesiodistal section of lower left first permanent molar showing remaining calcium hydroxide, decalcified dentine, and bulbous portion of pulp.

HISTOLOGIC SECTIONS

The mandibular left first permanent molar of a patient treated in the aforementioned manner was judged clinically and roentgenographically satisfactory after twelve months (Fig. 5). This tooth was extracted for orthodontic reasons and subjected to histologic sectioning. (This patient was not included in the clinical results of the study.)

Fig. 6 shows a vertical mesiodistal section of the lower left first permanent molar. The remains of the calcium hydroxide may be seen lying superiorly. The occlusal dentine shows a V-shaped area of decalcification with enlarged

tubules containing numerous microorganisms. Secondary dentine and predentine are forming on the pulpal surface of the occlusal dentine (Fig. 7). A definite zone of Weil is present, as well as an active odontoblastic layer.

The pulp contains some enlarged capillaries filled with red blood cells, and a slight hyperemic appearance is enhanced by the presence of inflammatory cells bordering the occlusal area of the pulp. The fibroblasts elsewhere in the pulp appear normal in size, slope, and distribution.

Fig. 7.—Enlargement of distal pulpal horn of lower left first permanent molar. Note secondary dentine, predentine, and zone of Weil. Pulp is slightly hyperemic, but fibroblasts are normal in size and distribution.

A deposition of secondary (irregular) dentine is seen on the pulpal side of the bifurcation (Fig. 6). On top of this is a calcified bulb of dentine with an active odontoblastic area and zone of Weil. This is thought to be an artifact peculiar to this tooth. The cementum appears normal throughout.

DISCUSSION

The final evaluation of the success of any dental treatment, specifically that involving the dental pulp, can be verified only by the patient's return to the dentist's office at specific intervals so that the outcome of treatment can be evaluated by predetermined criteria. The clinical results of other studies[1-3] in which patients have been treated and re-examined seem to indicate that presently accepted methods of treating deep carious lesions leave much to be desired. Although further investigation of the calcium hydroxide treatment of deep carious lesions is necessary, it would seem from the success of this study that the method has merit in selected clinical cases.

Several possible explanations may be advanced for treatment failure in the fourteen cases in this study. In some instances irreversible pulp degeneration with no outward detectable evidence may have already been in progress at the time of the initial appointment. Failure also could be attributed to mechanical loss of the soft amalgam filling material, either because of the weakness of the amalgam itself or because of the insufficient support afforded by the calcium hydroxide base.

We believe that both of these difficulties can be appreciably reduced. The diligent selection of cases should limit the application of this technique to only those cases in which carious lesions are known to be extensive, encroaching on the pulp but not actually penetrating the pulp chamber. The addition of a zinc oxyphosphate cement base over the calcium hydroxide before placement of the initial amalgam restoration would permit the amalgam to be placed in the usual manner, thus avoiding the possibility of fracture.

Upon removal of the calcium hydroxide and the remaining caries, if there was no pulp exposure, frequently a hard, homogeneous, semitransparent textured layer was found. Frequently the pulp could be seen beneath this surface. It was felt several times that a surgical exposure could have been made with a hand spoon instrument and slight pressure. One such slight exposure was made. The pulp was capped and the case was found to be a clinical success after two years of observation.

The cause of the radiopaque areas seen in the roentgenogram on the pulpal side of the caries is unknown.

SUMMARY

After two years, 76 per cent of all deciduous and young permanent teeth with deep carious lesions proved to be clinically and roentgenographically sound subsequent to procedures involving application of a calcium hydroxide paste over residual caries for a six-month period, followed by complete excavation and restoration by conventional methods.

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Section of the

FEDERAL DENTAL SERVICES

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NOMA

Report of a Case

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NOMA (gangrenous stomatitis, cancrum oris) is a rapid, ulcerating disease which generally occurs in debilitated persons.[1-3] It is highly progressive and destructive, and it affects chiefly the gingivae, cheeks, and lips but may spread to the ear, nose, eyelids, and external genital organs.[4]

Noma is predominantly a disease of childhood, particularly the decade between the ages of 2 and 12 years; most cases occur in children under 5 years of age.[5, 6] The incidence of this disease in the adult is universally uncommon. Although it affects both sexes, the disease seems to occur more often in females. Reports suggest that adults are affected especially in the third and fourth decades of life.[4]

Noma has a high fatality rate.

The specific cause of the disease is not known. Like ulcerative (Vincent's) stomatitis, noma is associated with the presence of large numbers of fusiform and spirochetal organisms.[1, 7]

There is widespread agreement that noma is rarely, if ever, a primary condition.[2] Outstanding among predisposing factors is malnutrition. Most authorities agree that noma seldom occurs in a well-nourished and well-cared for person.

A second factor is an unsanitary environment. Unsanitary home conditions and a markedly unhygienic mouth condition are usually found in conjunction with malnutrition.

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A third factor which is usually essential for the development of the lesion is a state of lowered resistance or debilitation. Noma usually occurs following attacks of measles, typhoid fever, scarlet fever, diphtheria, chronic malaria, smallpox, kala-azar, tuberculosis, bronchopneumonia, streptococcic septicemia, chronic nephrosis, dysentery, frank starvation, Vincent's angina, typhus, relapsing fever, erysipelas, leukemia, agranulocytosis, and enteritis.[2-4]

A fourth factor is trauma. Many reports indicate that there may be a relationship between the extraction of teeth and the subsequent point of commencement of the lesion.[1]

According to some authorities,[2] noma is not a disease in itself but a terminal sign and symptom of some debilitating disease.

Because noma is extremely rare in western countries, few cases have been reported in the literature. For this reason, the following report is presented.

CASE REPORT

On April 20, 1960, a 52-year-old Negro woman was brought by ambulance to the Emergency Clinic of Grady Memorial Hospital. Her main complaints were dysphagia, inability to eat solid foods due to lesions in her mouth, and general weakness.

Fig. 1.—Photograph showing extensive edema, trismus, and early necrosis of left cheek.

Examination.—Visual examination revealed a chronically ill, debilitated, and malnourished woman appearing much older than her stated age. At times she was disoriented while she was being questioned. Vitiligo was generalized. The left side of the face and neck was edematous, and there was periorbital edema. A black necrotic area (1 by 1 cm.) was seen on the left cheek (Fig. 1). The cervical lymph nodes on both sides were swollen, discrete, hard, and painful to palpation.

Intraorally, the left side of the mouth revealed extensive necrotic and gangrenous tissue extending from the orbicularis oris to the posterior pharyngeal wall and from the floor of

the mouth and tongue to the palate. Trismus was evident. Saliva drooled excessively from the mouth. An extremely fetid odor and marked destruction of tissue combined to produce a picture of offensive mutilation (Fig. 2). The temperature was 99° F., and the pulse rate was 90 beats per minute. Blood pressure and respiration were within normal limits.

The patient had fallen approximately four months prior to her appearance at the Emergency Clinic, and she had been bedridden since that time. Two weeks previous to her admission, she had a toothache in the left maxillary molar teeth, but no treatment was sought by her family. Since that time, the mouth condition had become progressively worse in that area. The patient was admitted to the hospital with a diagnosis of noma.

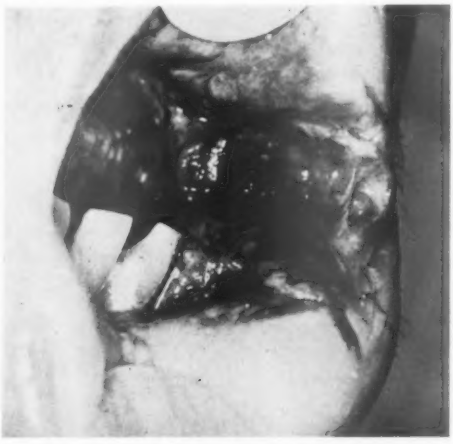

Fig. 2.—Photograph showing intraoral destruction.

Chest roentgenograms were negative. Blood studies on admission revealed a white blood count of 15,150. The hematocrit was 28 mm. The urinalysis was essentially negative except for a 2+ albumin. The urine had a specific gravity of 1.015. The serology was negative. Electrolyte studies of sodium, potassium, carbon dioxide, and chlorides were essentially negative.

Treatment.—The patient was immediately started on massive antibiotic therapy and intravenous fluids. Painful swallowing and trismus made it difficult for her to take fluids orally. She was given 20 million units of aqueous penicillin intravenously every eight hours and 1 Gm. of streptomycin intramuscularly every twelve hours. A 3 per cent solution of hydrogen peroxide was used to débride and clean the mouth every hour. Since the patient's hematocrit was low, she was given one unit of whole blood.

As the hours passed, the patient's condition became progressively worse and she became more disoriented. Death occurred forty-four hours after admission.

An autopsy was performed. The gross pathologic impressions were noma, left ventricular hypertrophy with minimal dilatation, cachexia, peritoneal and pericardial effusions, nutritional and/or cardiac cirrhosis, portal hypertension and chronic passive congestion of spleen, nephritis, nephrolithiasis, hemorrhagic cystitis, leiomyomas of the uterus, and solitary thyroid cyst.

SUMMARY

A case of noma is presented, since this condition is rare and few cases have been reported in the literature.

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Erratum

In the article by Amler and Johnson entitled "Transference of Osteogenic Tissue to the Anterior Eye Chamber by Means of Polyvinyl Plastic Sponge Grafts," which appeared in the July issue, the first United States Public Health Service research grant mentioned in the footnote on page 868 should have been No. 1065 instead of No. 6105.

EXTRAPYRAMIDAL SYNDROME DUE TO COMPAZINE THERAPY

Review of a Case

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A WHITE woman, 27 years of age, reported to the outpatient clinic of Offutt Air Force Base Hospital on Dec. 5, 1960, with the chief complaint of intermittent spasm and pain of the muscles of mastication. She was referred to the oral surgery service, where a provisional diagnosis of temporomandibular pain-dysfunction syndrome was ruled out.

Past History.—The past medical history was essentially negative and noncontributory. There was no history of allergies.

History of Present Illness.—On Dec. 1, 1960, when the patient was first seen in the outpatient clinic, her chief complaints were nausea and anxiety of one month's duration. Compazine, 15 mg. in the morning and at bedtime, was prescribed. On Dec. 4, 1960, the patient experienced blurred vision, vertigo, and fatigue. On Dec. 5, 1960, she reported to the hospital with pain and tonic spasm of the muscles of mastication.

Physical Examination.—The physical examination was essentially negative and noncontributory, with the exception of clinical evidence of tonic spasm of the muscles of mastication bilaterally. The severe intermittent spasms produced right and left lateral displacement of the mandible. Intraoral examination revealed areas of denudation of the buccal mucosa, bilaterally at the level of the retromolar regions. These denuded areas measured about 1.5 cm. in diameter.

Hospital Course.—The patient was admitted to the hospital, and 3.5 grains of Amytal sodium was administered intravenously. The muscle spasms were promptly relieved, and the patient could open and close her mouth normally and without discomfort. She was maintained on 30 mg. phenobarbital orally every six hours. Compazine therapy was discontinued. The patient was discharged from the hospital on the following day, and she has had no recurrence of tonic spasm or pain since.

DISCUSSION

Tranquilizing drugs are in very widespread use by the medical profession today. Following treatment with these drugs, patients often manifest extrapyramidal reactions or paradoxical behavioral reactions. There are two general types of reaction to the tranquilizing drugs:

1. Psychodynamically determined.
2. Neurologically determined.

1. *Psychodynamically determined reactions.* These occur when the drug removes or masks motor activity which the patient considers necessary for his psychological well-being. The patient, feeling that the drug is masking his

motor activity, will overact to satisfy his basic psychological need. An example of this is seen in the patient who is receiving a secondary gain from his illness, such as a pension, compensation, etc. When he sees that, as a result of treatment, he is losing the symptoms which make this gain possible, he will manifest bizarre responses to the drug in an effort not to lose the secondary gain.

2. *Neurologically determined extrapyramidal reactions.* These are induced by the tranquilizers, such as phenothiazine, perphenazine, trifluoperazine, chlorpromazine, and prochlorperazine. The extrapyramidal side effects are most commonly seen following administration of prochlorperazine, the essential substance of Compazine. These drugs act on the extrapyramidal areas of the nervous system, affecting the basal ganglion directly. The reactions usually fall into the following categories:

1. Parkinsonian-like reactions, characterized by gait and postural abnormalities with varying degrees of rigidity, tremor, and salivation.

2. Dyskinetic and dystonic syndromes, characterized by spasms or cramps of several muscle groups. Syndromes involving the face and neck are most common. The patient manifests pain and inability to open the mouth. These symptoms are best treated by discontinuing the drug and administering a barbiturate, the drug of choice being Amytal sodium, 3.5 to 7.5 grains intravenously.

SUMMARY

An increasingly large number of persons are being treated with tranquilizing drugs. It is quite likely that a patient having a reaction to a tranquilizer which involves trigeminal nuclei will report to a dentist's office, thinking that his problem is of "dental origin." The case described here was unusual in that only the trigeminal nuclei bilaterally were involved, producing the extrapyramidal side effect. As a result of this experience, we believe it most prudent to get an adequate history with respect to medication which the patient may be taking and thereby arrive at an early correct diagnosis and a proper treatment plan. It seems most reasonable to us that, in the case we have presented, the fact that the patient had been taking the medication in relatively small doses for a short time precludes the diagnosis of a psychodynamic reaction. Therefore, we conclude that the manifestation of this extrapyramidal syndrome was on a neurological basis entirely due to Compazine therapy.

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ANNOUNCEMENTS

American Academy of Implant Dentures

The Academy will meet Oct. 13 to 15, 1961, at the Dental School of the University of Pennsylvania in Philadelphia, just prior to the meeting of the American Dental Association. The program follows:

Closed-Circuit Demonstrations of Implant Procedures.
Drs. Goldberg and Gershkoff and Drs. Leonard and Jermyn.

Lectures on Implant Techniques. Frank Strake, Norman Cranin, Isaiah Lew, Eric Bausch, and Academy members from the United States and abroad.

Didactic Lectures by faculty members of the University of Pennsylvania:

1. Dean Lester Burket.
2. LeRoy Ennis: Roentgenographic Techniques and Interpretation of Edentulous Jaws.
3. H. Milton Rode: A Prosthodontist Comments on the Implant Denture.
4. Claude LaDow: Oral Surgical Techniques.
5. Leon Collins: The Medical Responsibilities and Rewards of the Oral Surgeon.

The tuition fee is $50.00. Further information may be obtained from Dr. A. Norman Cranin, 2120 Ocean Ave., Brooklyn 29, New York.

Dental Guidance Council for Cerebral Palsy

The annual scientific meeting of the Dental Guidance Council for Cerebral Palsy will be held at the Academy of Medicine Building, 103rd St. and Fifth Ave., New York City, on Thursday, Nov. 16, 1961, at 8:30 P.M. The title of the symposium is "The Oral-Facial Complex in Patients With Neuromuscular Disorders." Andrew M. Linz, D.D.S., M.Sc. (Dent.), president of the Council, is chairman of the meeting. Panelists are Ross M. Campbell, M.D., Mary Stewart, M.A., and Albert Green, D.D.S.

Further information may be secured at the Council's offices at 70 Fifth Ave., New York 11, New York.

The Council's officers for 1961-1962 are as follows:

Honorary President—James Jay
President—Andrew M. Linz
President-Elect—Albert Green
Vice-President—Marvin King
Secretary—Herbert Hayward

Hospital Dental Chiefs Form National Association

The first national organization conference of the American Association of Hospital Dental Chiefs will be held at the Philadelphia General Hospital in Philadelphia, Pennsylvania, on Oct. 16, 1961. The meeting will begin at 4:30 P.M.

Philadelphia Endodontic Study Club

An all-day endodontic program will be presented by the Philadelphia Endodontic Study Club on Sunday, Oct. 15, 1961, from 9:30 A.M. to 4:30 P.M. At the morning session papers on "Clinical Endodontic Research" will be read. A panel discussion of "The Control of Pain in Endodontics" will be presented in the afternoon.

A check to cover the $15.00 registration fee (which includes luncheon) should be sent to Dr. Morris Zitaner, 7244 Frankford Ave., Philadelphia, Pennsylvania.

Workshop on Adhesive Restorative Materials

The National Institute of Dental Research of the United States Public Health Service has announced that a grant of $9,600.00 has been awarded to Indiana University School of Dentistry to organize an initial workshop on the development of an adhesive restorative material. The chairman of this workshop will be Ralph W. Phillips, and the co-chairman will be Gunnar Ryge. The workshop will be held in Indianapolis, Indiana, on Sept. 28 and 29, 1961. The entire proceedings from this two-day workshop will be transcribed and published.

University of Alabama

The School of Dentistry will offer the following courses during the months of October and November, 1961:

Dental Assisting. Oct. 28 and 29, 1961. Paul E. Hammons and Gertrude M. Sinnett, assisted by other faculty members.

Complete Dentures. Nov. 4 to 6, 1961. John J. Sharry.

Clinical Oral Surgery. Nov. 11 to 13, 1961. Colonel Robert B. Shira.

Root Canal Therapy. Nov. 25 to 27, 1961. Adeeb E. Thomas.

Further information regarding these programs can be obtained by writing Director, Refresher Course Program, University of Alabama School of Dentistry, 1919 Seventh Ave. South, Birmingham, Alabama.

Morrisania City Hospital

A surgical internship, accredited by the American Dental Association, is available for 1962 at Morrisania City Hospital, Bronx, New York. Complete information may be obtained by writing to the Hospital Medical Superintendent.

Mount Sinai Hospital

The Department of Dental and Oral Surgery will offer the following postgraduate courses under the direction of Dr. Alvin S. Nathan:

1. Analgesia and Pain Control in Dental Practice. Oct. 6, 13, 20, and 27, 1961, 2 to 6 P.M. Alvin Nathan, William Greenfield, Walter Caruso, and Norman Cranin. Tuition, $60.00.
2. Minor Oral Surgery for the General Practitioner. Nov. 3, 10, 17, 24, Dec. 1, 1961, 2 to 6 P.M. Alvin Nathan, L. Stern, Jr., Marvin Freid, Peter Stern, N. Cranin, and members of oral surgery staff. Tuition, $65.00.

Information, brochures, and application forms may be obtained by writing Dr. A. Norman Cranin, Chairman, Education Committee, Department of Dentistry, The Mount Sinai Hospital, 100th St. and Fifth Ave., New York 29, New York.

Temple University

An intensive participation course in clinical endodontics will be presented at the School of Dentistry Nov. 6 to 11, 1961, under the direction of Dr. Leonard Parris. This course will fulfill the prerequisite requirements for the postgraduate course entitled "Surgical Endodontics" to be presented in April, 1962.

For further information and application, write to Dr. Louis Herman, Director of Postgraduate Studies, Temple University School of Dentistry, 3223 North Broad St., Philadelphia 40, Pennsylvania.

Registry of Gingival Disease Established at Tufts University

The increasing awareness of the importance of gingival disease has created the need for a clearing house for the histopathologic evaluation of diagnostic and treatment problems. For that reason, the Department of Periodontology of the Tufts University School of Dental Medicine announces the establishment of a Registry of Gingival Disease. Practitioners are invited to submit gingival biopsy material for diagnosis. Additional pertinent information regarding the nature of individual cases will be provided by the Registry. Permanent files will be maintained for follow-up case study. Necessary forms and mailing containers will be sent on request. There is no charge for this service.

Interested persons may write to the Department of Periodontology, Tufts University School of Dental Medicine, 136 Harrison Ave., Boston 11, Massachusetts.

Tufts University

The School of Dental Medicine announces the following postgraduate refresher courses:

201 A. Cancer of the Oral Cavity and Salivary Glands. Six Thursdays, beginning Oct. 12, 1961, 1 to 4 P.M. Gerald Shklar, Irving Meyer, and staff. Tuition, $125.00.

301 A. Hospital Management of the Dental Patient. Feb. 7 to 9, 1962. Richard Taylor and Dental Department of Boston City Hospital. Tuition, $100.00.

300 A. A Participation Course in Applied Anatomy of the Head and Neck. Feb. 28 to March 2, 1962. Alton D. Brashear and Dental Department of Boston City Hospital. Tuition, $100.00.

202 A. The Diagnosis, Management and Therapy of Lesions of the Oral Mucosa. March 7 to April 25, 1962 (eight consecutive Wednesdays). Philip McCarthy, Francis P. McCarthy, and Gerald Shklar. Tuition, $125.00.

200 A. Surgical Pathology of the Mouth and Jaws. March 8 to May 10, 1962 (eight Thursdays), 1 to 4 P.M. Irving Meyer, Gerald Shklar, and staff. Tuition, $125.00.

302 A. Minor Oral Surgery. May 8 to 10, 1962. Richard Taylor and staff, Boston City Hospital. Tuition, $125.00.

For further information concerning these courses, please write to Dr. L. Walter Brown, Jr., Tufts University School of Dental Medicine, 136 Harrison Ave., Boston 11, Massachusetts.

Tufts University Announces Periodontology Curriculum

The curriculum in periodontology has been developed with a view toward the expanding interrelationships of modern periodontal practice with other phases of dentistry, medicine, and biologic science.

Periodontology I is a two-year (two academic years of eighteen consecutive months) specialist-training program for persons interested in qualifying for certification in periodontia.

Periodontology II, a one-year specialist-training program, is offered for persons with one year's previous graduate or postgraduate training in periodontology.

Periodontology III is intended for persons interested in the academic and research aspects of periodontology, as well as preparation for specialty board certification. The course ordinarily takes two years and leads to the degree of Master of Science or Master of Dental Science.

For further information write: Director, Division of Graduate Studies Tufts University School of Dental Medicine, 136 Harrison Ave., Boston 11, Massachusetts.

Boston University

The following postgraduate courses are offered by the Department of Stomatology, School of Medicine, Boston University and the Massachusetts Memorial Hospitals:

Occlusal Adjustment. Oct. 30 to Nov. 3, 1961. Drs. Bernard S. Chaikin, Henry M. Goldman, Gerald M. Kramer, William Pendergast, Irving Romanow, J. David Kohn, David J. Baraban, Leo Talkov, David V. Berry, and Jack Bloom. Fee, $200.00.

Periodontal Therapy. Nov. 6 to 10, 1961. Drs. Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, William Pendergast, Irving Romanow, Jack Bloom, and associates. Fee, $200.00.

Endodontics. Nov. 13 to 17, 1961. Dr. Herbert Schilder. Fee, $175.00.

Fixed Bridge Prosthesis. Nov. 27 to Dec. 1, 1961. Drs. Leo Talkov, David J. Baraban, and Lloyd Warshauer. Fee, $175.00.

Periodontal Pathology. Jan. 22 to 26, 1962. Drs. Henry M. Goldman and Jack Bloom. Fee, $200.00.

The Use of Ultrasonics in Periodontal Therapy. March 17, 1962. Dr. Henry M. Goldman. Fee, $35.00.

Periodontal Therapy. March 19 to 23, 1962. Drs. Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, William Pendergast, Jack Bloom, and associates. Fee, $200.00.

Advanced Course in Periodontal Therapy. March 26-30, 1962. Drs. Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, William Pendergast, and associates. Fee, $200.00.

Apicoectomy—Surgical Endodontic Procedures. April 2 to 6, 1962. Dr. Herbert Schilder. Fee, $175.00.

Treatment Planning Clinic—Restoration of Missing Teeth. April 6 and 7, 1962. Staff, Department of Stomatology. Fee, $60.00.

Surgical Procedures in Periodontal Therapy. April 30 to May 4, 1962. Dr. Lewis Fox and associates. Fee, $200.00.

Full Mouth Reconstruction. May 14 to 18, 1962. Drs. Leo Talkov, David J. Baraban (Restorative Phase), Henry M. Goldman, Bernard S. Chaikin, Gerald M. Kramer, J. David Kohn (Periodontal Phase). Fee, $200.00.

For applications, write to David J. Baraban, D.M.D., Director of Postgraduate Studies, Department of Stomatology, Massachusetts Memorial Hospitals, 750 Harrison Ave., Boston 18, Massachusetts.

American Academy of Periodontology

The Academy will hold its forty-seventh annual meeting aboard the *M. S. Bergensfjord*, sailing from Philadelphia to Bermuda on Oct. 20 and returning to Philadelphia on Oct. 25, 1961. In Bermuda the ship will serve as the Academy's hotel.

Any member of the American Dental Association or recognized affiliate organizations may secure space on the *M. S. Bergensfjord* as long as space is available.

Those interested in participating in this meeting should contact Mr. E, M. Baskette, Caribbean Cruise Lines, Suite #301, Roeper Bldg., Miami 32, Florida.

American Academy of Oral Roentgenology

The annual scientific meeting of the Academy will be held at the Warwick Hotel in Philadelphia, Pennsylvania, on Saturday, Oct. 14, 1961. This meeting will be preceded on October 13 by an open Executive Committee meeting at which the affairs of the Academy will be discussed.

Mid-Continent Dental Congress

The Mid-Continent Dental Congress will be held Oct. 29 to Nov. 1, 1961, at the Chase Hotel in St. Louis, Missouri. The following papers will be presented:

Monday, October 30

Some Aspects of the Dental Research Program at the National Bureau of Standards. George C. Paffenbarger, D.D.S., Washington, D.C.

X-ray Diagnosis in the Dental Office. William E. Koch, Jr., D.D.S., St. Louis, Missouri.

Call the Next Witness for the Defense—Legal Aspects of Dentistry. Richard F. Rabe, D.D.S., J.D., Des Moines, Iowa.

Conservation of Tooth Substance in the Preparation of Bridge Abutments. Roland W. Dykema, D.D.S., Indianapolis, Indiana.

Tuesday, October 31

Periodontia: A Part of Every Practice. Lloyd J. Phillips, D.D.S., Indianapolis, Indiana.

Treatment of Emergencies in the Dental Office. Robert E. Allen, D.D.S., M.S., Kansas City, Kansas.

The Problem of the Loose, Uncomfortable Denture. Bernard Jankelson, D.M.D., Seattle, Washington.

Wednesday, November 1

The Control of Pain in the Dental Office. Norman H. Olsen, D.D.S., M.S.D., Chicago, Illinois.

Communication—The Secret to Successful Human Relations. Stanleigh B. McDonald, B.S., Indianapolis, Indiana.

For complete details, write to Greater St. Louis Dental Society, 8013 Maryland Ave., Clayton 5, Missouri.

American Dental Society of Anesthesiology

The Society will hold its annual meeting on Oct. 13, 14, and 15, 1961, at the Statler-Hilton Hotel, New York, New York.

REVIEWS OF THE LITERATURE

BOOK REVIEWS

Synopsis of Oral Pathology. S. N. Bhaskar, B.D.S., D.D.S., M.S., Ph.D., Major, United States Army Dental Corps; Chief, Oral Tumors Branch, Armed Forces Institute of Pathology, Washington, D. C.; Assistant Professor of Pathology, Georgetown University School of Dentistry, Washington, D. C.; Lecturer, Army Medical Service Graduate School, Walter Reed Army Medical Center, Washington, D. C.; Consultant in Oral Pathology to the Assistant Surgeon General and Chief of the Dental Corps, United States Army; Member, Dental Research Advisory Committee to the Medical Research and Development Division, Department of the Army; formerly Associate Professor of Pathology, University of Illinois College of Dentistry, Chicago, Illinois. St. Louis, 1961, The C. V. Mosby Company. 502 pages, 325 illustrations. Price, $9.75.

The author has given to the dental profession a valuable diagnostic aid which utilizes the abundant material at the Armed Forces Institute of Pathology. Part I of this book is composed of a comprehensive classification of lesions according to their clinical features. The classification is an excellent ready reference source of information for the student or busy practitioner. Brief descriptions of the characteristics, prognosis, and treatment of a particular lesion are given, along with the corresponding page number for a more detailed description.

Part II is composed of a condensation of the various subdivisions of oral and dental pathology. The descriptions are concise, yet factual and thorough, and there are numerous exceptional illustrations. The handy pocketbook size is appealing. The book is well organized, and the author does not elaborate on microscopic detail but presents clinical manifestations of oral disease. Dr. Bhaskar has given us a valuable addition to our library.

Richard T. Gaines.

Concepts of Medicine: A Collection of Essays on Aspects of Medicine. Brandon Lush, M.D., M.R.C.P. New York, 1961, Pergamon Press, Inc. Price, $8.50.

Associated with the editor of this interesting book are many outstanding men of medicine.

Great sageness is expressed in the many essays. Platt, in the first essay, writes: "Some clinical problems can be solved by knowledge, some by experience and some by understanding. The application of these three qualities has come to be known as clinical instinct or clinical wisdom."

Allen, near the end of the book, writes: "Medical research has but one goal, the understanding and relief of illness of the human being in general. The talents of the investigator in medicine will flower fully only when he understands the needs of patients."

It is a delightful book, which makes one think of himself. As so well expressed by Bush: "In the first place let us record that there are individuals, members of every profession, who will never collaborate with anyone under any circumstances. Let them depart in peace: their day is nearly done."

I will read this book many times over; once was not enough.

Thomas J. Cook.

ABSTRACTS OF CURRENT LITERATURE

Complications of Tracheostomy. O. J. King, Jr., and W. W. Glas. Rocky Mountain M. J. **67:** 36, August, 1960.

The purpose of this investigation was to determine (1) the complications of tracheostomy, (2) their frequency, and (3) methods of preventing these complications.

A review of tracheostomies performed at the authors' hospital showed that 19 complications occurred in 78 cases in the past year. These complications are listed as hemorrhage at the operative site (7 cases), recurrent respiratory obstruction (4 cases), ulcerative tracheobronchitis (3 cases), pneumothorax (2 cases), tracheo-esophageal fistula (2 cases), and apnea, shock, and death (1 case).

Complications developed in 42 per cent of those patients in whom tracheostomies were performed under emergency conditions. Only 19 per cent developed complications when the operation was performed as an elective procedure.

It is explained that the most frequent complication, hemorrhage, is due largely to the fact that tracheostomies were often performed in patients who were hypotensive. Small vessels were not evident and hence were not ligated. With supportive care, the blood pressure increased and these vessels began to bleed.

The authors offer an explanation for the puzzling syndrome of apnea, shock, and death following tracheostomy.

Edmund I. Parnes.

Antibiotics in Surgical Cases. E. J. Pulaski. Arch. Surg. **82:** 545, April, 1961.

Antibiotics are used to control or eliminate bacterial infections and to prevent complications following surgical procedures in or about the oral cavity.

New antibiotics add new responsibility, for until they are tried there is danger of their having an inherent toxicity and of possible sensitization and other reactions in individual patients.

Antibiotics are not all unique and distinct in their activities. It is good practice to diagnose infection, identify the causal bacteria, and determine the antibiotic effective against these bacteria.

Although there is considerable overlapping, certain antibiotics have proved helpful in their action against certain bacteria, as follows:

Gram-positive organisms—Penicillin, erythromycin, novobiocin, bacitracin, ristocetin, vancomycin, tyrothricin, leucomycin

Gram-negative organisms—Streptomycin, polymyxins, colistin, paromomycin

Gram-positive and gram-negative organisms—Tetracyclines, chloramphenicols, neomycins, kanamycin

Fungi—Nystatin, amphotericin B, griseofulvin

The use of any systemic antibiotic involves a calculated risk. An antibiotic should be administered only in the presence of an infection which is likely to respond to it. Some of the hazards are common, others rare; some depend on dosage, others on the period of treatment.

The prophylactic use of antibiotics is considered a misuse of the drug. A careful history of the patient's past behavior with respect to an antibiotic is important before administration of the drug.

This excellent article is recommended to the oral surgeon.

T. J. C.

Conference on Intraoral Cancer. N. A. McCormick. Canad. M. A. J. **83:** 152, July, 1960.

From 1938 to 1952 1,548 cases of intraoral cancer were registered at the Ontario Regional Cancer Clinics. This figure represents 2.7 per cent of all new cases of cancer registered.

Cigar and pipe smoking and tobacco chewing were found to be contributing factors. Excessive use of alcohol, poor dental hygiene, and nutritional deficiencies all play a part and must be corrected if treatment is to be successful.

Intraoral cancer should be treated by irradiation, with surgical treatment reserved for complications resulting from infection or tumor invasion of nearby bony structures. In this series 1,009 patients received no treatment to the neck except as the neck was included in the field of external irradiation.

Oral sepsis must be controlled, since it reduces the response to irradiation and increases the immediate dangers of necrosis and sequestration from any form of treatment in which bone is involved.

Three interesting case reports illustrating the treatment of intraoral carcinoma are presented.

T. J. C.

A Statistical Review of Intraoral Cancer. A. H. Sellers. Canad. M. A. J. **83:** 156, July, 1960.

Each year intraoral cancer is the underlying cause of the deaths of thirty-two male and seven female residents of Ontario, Quebec. Of 1,548 cases of intraoral cancer reviewed, 1,389 were microscopically confirmed as malignant.

The intraoral sites were as follows:

Buccal mucosa—570 cases
Floor of the mouth—266 cases
Inferior alveolus—291 cases
Superior alveolus—166 cases
Alveolus unspecified—41 cases
Hard palate—82 cases
Soft palate—63 cases
Palate unspecified—18 cases
Other or combined sites—51 cases

The first symptom was usually a lump or swelling, pain, soreness, or tenderness in the mouth, or a sore or ulcer. In ninety-four cases the earliest symptom was leukoplakia. The largest percentage of patients received irradiation treatment.

The median survival time in all confirmed cases of primary oral cancer was 28.6 months.

T. J. C.

Interosseous Wiring of Mandibular Fractures: A Follow-up of Fifty Cases. Anthony Ditchfield. Brit. J. Plast. Surg. **13:** 146, July, 1960.

During a recent six-year period, 86 (15 per cent) of a total of 570 cases of mandibular fracture seen at the Plastic and Jaw Department of the United Sheffield Hospitals were treated by interosseous wiring. Of these, 50 were traced at least one year after injury.

The author lists many significant results and conclusions from his study.

In all fifty cases bony union was achieved; occlusion was restored in all cases; twenty-eight scars were considered inconspicuous; no anesthesia of the lower lip was presented, although twenty-two patients had paresthesia.

Complications can be anticipated under certain circumstances, such as a delay in operation, a poor skin cover over the wire, fragmentation of the alveolus and a number of teeth resulting in a large communication between the mouth and the fracture, loose fragments of bone, inefficient immobilization, and poor dentition.

Edmund I. Parnes.

Closed-Chest Cardiac Massage, Clinical Anesthesia Conference. New York J. Med. 61: July 1, 1961.

Closed-chest cardiac massage may revolutionize cardiac resuscitation techniques, and when combined with mouth-to-mouth ventilation it enables anyone anywhere to initiate complete resuscitation.

A case of successful resuscitation in a 29-year-old woman admitted to the hospital for excision of residual tonsillar tags is presented in the interest of re-emphasizing the basic contribution of Kouwenhouen, Jude, and Knickerbocker.

"Closed-chest massage probably should be the preferred approach to cardiac resuscitation outside the hospital and in the hospital other than in the operating room." If with each sternal compression a palpable peripheral pulse is obtained, there is no question that the massage is successful.

The full acceptance of the technique of closed-chest massage must depend upon reports of successful resuscitations from many groups.

Edmund I. Parnes.

Fracture-Dislocations of Zygoma and Zygomatic Arch. Howard W. Smith and Ei ji Yanagisawa. A. M. A. Arch. Otolaryng. **73:** 2, 1961.

This article discusses the historical development of surgical treatment of fractures of the zygoma and zygomatic arch. During the past 200 years at least twenty-three methods and twenty-four instruments have been introduced. In the past ten years at least one new method was recorded annually.

"The past 50 years have seen a change in the injury itself. Most zygomatic fractures are now part of a number of concomitant facial bone fractures, and therefore procedures which have a place in the over-all management of facial injuries have gained favor."

Edmund I. Parnes.